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SOME ASPECTS OF THE PATHOLOGY OF BONE TUMOURS OF INTEREST TO RADIOLOGISTS.[1]

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IT is customary in discussing tumours of the skeleton to deal first with primary growths and then with secondary growths. I propose to reverse that order and to discuss secondary tumours first. My reasons for doing this are three: (i) secondary tumours are much more common than primary malignant tumours of bones; (ii) metastatic growths from symptomless unsuspected primary tumours elsewhere have often been mistaken for primary bone tumours or other primary bone diseases both by clinicians and radiologists; and (iii) an appreciation of some of the incidental structural features seen in metastatic growths, especially the formation of new bone by expanded periosteum, is of great importance in the interpretation of skiagraphic appearances. I shall therefore dwell on these three points before proceeding to discuss primary neoplasms of bones.

[1] A lecture delivered before the Radiological Section of the Victorian Branch of the British Medical Association on June 21, 1938.

Metastatic Tumours in Bones.

Frequency of Occurrence.

The frequency of occurrence of metastatic tumours in bones exceeds that of primary tumours. An approximate estimate, though of course not statistically accurate, of the relative frequency of secondary and primary malignant tumours of bone,

is afforded by the following figures. I performed 500 consecutive autopsies in cases of malignant disease of all kinds. This series included the lesions mentioned in Table I.

Table I.

Metastatic Tumours in Bones.		Primary Tumours in Bones.	
Type of Tumour.	Number of Subjects.	Type of Tumour.	Number of Subjects.
Carcinoma	60	Sarcoma	5
Sarcoma	6	Sacral chordoma ..	2
Neuroblastoma	2	Myelomatosis	1
Melanoma	1		
Total	69	Total	8

Thus, in my *post mortem* material, subjects with metastatic growths in bones were over eight times as numerous as subjects with primary malignant tumours; and when it is remembered that each of the former cases has, on an average, many different metastatic tumours in various bones, it will be realized that individual metastatic growths outnumber individual primary growths probably several hundred times. The important fact emerges that, while secondary tumours in bones are very common, primary malignant tumours are quite rare.

Similarity to Primary Bone Disease.

The fact that metastatic growths often simulate primary bone disease is one which I have stressed elsewhere (Willis, 1931 and 1934, and Trinca and Willis, 1936), but which cannot be too often repeated. I can recall many cases in which, even after highly competent and prolonged clinical and radiological study, secondary tumours in bone have been thought to be some primary skeletal disease. I have seen metastatic growths diagnosed as "osteogenic sarcoma", "Ewing's tumour", "spinal tuberculosis", "cystic disease of bone", "chronic osteitis" and "chronic spondylitis". Space will not permit of details of the various cases, some of which have already been recorded in the papers just referred to. The following example, however, has not been reported previously.

E.J. (*post mortem* reference number 422), a man, aged seventy-five years, was admitted to hospital with the diagnosis of cervical osteoarthritis. He had suffered from persistent pain in the back of the head and neck for three months. Skiagrams revealed prominent lipping of many vertebræ, both in the cervical and lumbo-dorsal regions; and areas of irregular density seen in the vertebral shadows were interpreted as being due to osteoarthritis. Except for general emaciation and crepitations audible at the bases of both lungs, examination of the thorax and abdomen revealed no abnormality. The patient died a few days later. Autopsy disclosed a small ulcerated carcinoma of the stomach with deposits in the lymph glands of the lesser curvature, and multiple metastatic growths in the liver, both adrenal glands and the cervical and lumbar vertebræ and the base of the skull. In a review of the skiagrams some of the diseased areas in the vertebræ could be identified as corresponding to the growths seen *post mortem*; but the prominent osteoarthritic changes which were also present had masked the rarefactions due to the growths.

Such cases show that metastatic growths can and do elude discovery by careful and competent workers; and this should engender great caution in pronouncing as "primary" any obscure skeletal lesion. The radiologist should always ask himself: Could this possibly be some metastatic growth?

New Bone Formation in Metastatic Tumours.

All radiologists and pathologists are familiar with the process of osteoplasia in bones which are the seat of metastases from carcinomata of the prostate, breast or (rarely) other organs. The affected bone becomes denser, heavier and more radio-opaque than normal, and osteophytic outgrowths may spring from its surface. The invading tumour in some way stimulates proliferation in the bony stroma in which it is growing. It is important to realize that a tumour which provokes osteoplasia in one place may cause osteolysis or rarefaction in another place; and it is even more important to appreciate that a metastatic growth may cause neither osteoplasia nor osteolysis, but may merely occupy marrow spaces and cause no significant change in the bony trabeculæ. Skiagraphy will, of course, fail to reveal any alteration of bony architecture in such a growth, which indeed will be invisible and undiagnosable by this means. On several occasions I have seen at autopsy obvious areas of white growth in vertebræ which, however, had suffered neither sclerosis nor rarefaction, and which therefore appeared in skiagrams to be substantially normal.

Now, in addition to the well-known process of osteoplasia, there is another kind of new bone formation in metastatic tumours which is less widely known. This is the formation of bone by a periosteum which is suffering elevation from the bone surface and expansion by an enlarging subperiosteal growth. In these circumstances the new bone may be laid down by the affected periosteum in either of two ways: (*a*) as radially projecting brush-like spines arranged more or less vertically to the surface of the disease bone, or (*b*) as thin laminæ or "onion-skin" layers arranged parallel to the surface of the bone (Figure I). This periosteal activity may be seen so prominently in a tumour which itself has no bone-forming powers, that the growth may be thought erroneously to be osteogenic.

The two types of arrangement of the periosteal new bone, namely, the radial spines and the parallel laminæ, may be seen in different parts of the same growth. Good specimens of both are afforded by the metastases of neuroblastomata of the adrenal glands or sympathetic ganglia.

Figure II depicts part of the skiagram of the femur of a girl, aged eight years, whose history has been recorded by Colville and myself (1933). The tumour, which was regarded clinically as a Ewing's tumour of the femur, was shown at autopsy to be a metastasis from an adrenal neuroblastoma. The skiagram shows clearly the "onion-skin" layers of new bone deposited by the elevated periosteum.

Figure III is a skiagram of a small part of the vault of the skull from a child, two years old, who died from

an adrenal ganglioneuroma with widespread neuroblastomatous metastases in lymph glands, liver and many bones. These last included large bosses of growth on the skull, constituting Hutchison's syndrome. As the skiagram shows, the tumour bosses on both the inner and outer aspects of the skull contain many radially arranged spike-like projections of new bone, which have been laid down as a result of the activity of the elevated periosteum. In some of the long bones in the same case skiagrams obtained during life revealed "onion-skin" layers. The radiologist remarked that they resembled those of Ewing's tumour.

Figure IV is an enlarged photograph of a section of a rib from another child, aged four years, who died from a large retroperitoneal ganglioneuroma with widespread neuroblastomatous metastases in many viscera and bones. All bone visible in the photograph has been blacked in, so that we obtain a clear silhouette of the section of the rib and of the bony spicules projecting from it into the subperiosteal growth.

Now, while it is true that neuroblastomata often provide beautiful specimens of both laminated and radial types of periosteal formation of new bone, there is good reason for believing that these appearances are not diagnostic of any particular tumour. Brunschwig and Harmon (1935) have shown experimentally in rats that radiating spicules of bone may develop as a result of the subperiosteal growth of a soft-tissue sarcoma; and in a thoughtful and well-reasoned paper they discuss the rôle played by the periosteum in various human tumours.

Osteogenic Sarcoma.

With the foregoing points regarding secondary tumours well in mind, let us now turn attention to primary tumours of bones, beginning with the osteogenic sarcoma.

Since non-osteogenic secondary tumours in bones may be accompanied by the periosteal formation of new bone, may not the same apply to sarcoma of bone itself? May not much of the new bone seen in osteogenic sarcomata arise, not by differentiation in the tumour tissue, but through the activity of the elevated periosteum? Brunschwig and Harmon contend that this is indeed the case, and that the radiating spiny "skeleton" seen in some osteogenic sarcomata is of periosteal origin and is exactly comparable with that seen in non-osteogenic secondary growths. From my own observations, I am satisfied that this view is correct. Consider, for example, Figures V and VI, obtained from a case proved *post mortem* to be of osteogenic sarcoma of the femur. The subject was a boy, aged fourteen years. In the skiagram, the newly formed bone which is visible in the tumour consists partly of a layer corresponding in position to the expanded periosteum and partly of coarse, irregular, radially arranged masses. In the microscopic section it is seen that these radial masses consist of well-formed bony spurs orientated vertically to the periosteal layer which encloses the margins of the tumour. The tumour tissue itself displays some osseous differentiation; but this is very patchy and incomplete and is represented mainly by uncalcified young osteoid tissue. It is clear that the bulk of the well-differentiated bone in the growth, comprising the periosteal layer and the radially arranged spurs, is the product not of the tumour itself but of the elevated periosteum which ensheathes it.

If, then, the visible ossification in a sarcoma of bone is often of non-neoplastic periosteal nature, what criteria are left for the skiagraphic diagnosis of primary osteogenic sarcoma? Massive and widespread ossification, neither laminated nor radial in position, will, of course, usually make the diagnosis clear; but when the ossification is scanty and principally of radial or laminated distribution, the distinction from a secondary tumour will often be difficult or impossible on purely skiagraphic grounds.

In cases difficult of diagnosis skiagraphically, there are five other points which may aid in diagnosis. First, the age of the patient is of significance. The victims of osteogenic sarcoma are quite commonly children or adolescents, in whom, of course, carcinomatous metastases in bones are exceedingly rare. Metastatic neuroblastoma, however, occurs almost exclusively in childhood or adolescence. Hence, a malignant bone tumour in a patient aged less than, say, twenty-five years, is much more likely to be either osteogenic sarcoma or metastatic neuroblastoma than metastatic carcinoma; but in a middle-aged or elderly patient it is very likely to be a metastatic carcinoma.

Secondly, the situation of the tumour is important. The sites of metastatic carcinoma in the skeleton correspond closely with those of red bone marrow; that is to say, the bones commonly affected are the axial and trunk bones and only the upper parts of the proximal segments of the limbs, the femur and the humerus (Willis, 1934). Except in cases of very widespread metastatic disease of the skeleton, in which, of course, difficulty in diagnosis is not likely to arise, metastatic carcinomata of the more distal bones of the limbs, though they occasionally occur, are rare. A malignant tumour of skiagraphically doubtful nature in the lower end of the humerus or femur, or in the radius, ulna, tibia, fibula or bones of the hands or feet, is more likely to be a primary one than to be a metastatic carcinoma. It is, however, only a question of likelihood; exceptions will occur.

Thirdly, bone formation in metastases may sometimes be seen in cases of osteogenic sarcoma (Willis, 1934, Brunschwig and Harmon, 1933, and Harding and Conrville, 1934). It will be only on rare occasions that this will prove useful diagnostically; but the presence of skiagraphically visible ossification in the pulmonary metastases accompanying a bone tumour (as in Figure VII) would prove almost conclusively the osteogenic nature of the tumour.

Fourthly, rarely, associated bone disease may point to a diagnosis of osteogenic sarcoma. Thus it is well known that a bone affected by Paget's disease may become sarcomatous. Hence, a malignant bone tumour with new bone formation arising in a patient with *osteitis deformans* is more likely to be a sarcoma than a metastatic carcinoma;

though, of course, people with this disease are not exempt from carcinoma. Figure VIII shows a sarcoma of the scapula in a case of Paget's disease.

Fifthly, estimation of the blood phosphatase may come to be a useful diagnostic test for osteogenic sarcoma. Phosphatase is a ferment produced by osteoblasts, and Franseen and McLean (1935) find that the amount of the ferment present in the blood is raised to a high level in cases of osteogenic sarcoma, a level usually higher than in any other bone disease, either primary or secondary. Such a test may prove of value in the diagnosis of bone tumours when the skiagraphic and other evidence is inconclusive.

Non-Osteogenic Solitary Tumours of Bones.

The most important of the non-osteogenic solitary tumours of bones is the so-called benign giant-celled tumour (giant-celled myeloma or myeloid sarcoma). We need not dwell on the well-known skiagraphic and pathological characters of this tumour; but it is interesting to note that the evolution of our knowledge of its behaviour is epitomized in its nomenclature. The old name of myeloid sarcoma was applied to it because of its cellular, vascular, malignant-looking microscopic structure. Gradually it began to be realized, however, that in spite of their structure these growths were easily eradicable, did not often recur after adequate local removal, and were not disseminated and did not kill. It then became the fashion to speak of them as benign giant-celled tumours of bone. Now, however, we know that that name also is inappropriate, since many cases have recently been reported in which tumours clinically, skiagraphically and histologically typical of this class have produced metastases and destroyed the patient (see King, 1932, Korchow, 1933, Willis, 1934). The title "myeloid sarcoma" is, then, not unjustified; and it serves to remind us that the benignancy so ardently insisted on by Bloodgood and others is far from absolute.

Solitary soft-tissue sarcomata, other than the giant-celled tumour, occasionally arise centrally in bones, sometimes causing the bone to expand and producing skiagraphic appearances not unlike those of the giant-cell tumour. These include fibrosarcomata, liposarcomata and neurogenic sarcomata. An example of a central fibrosarcoma is depicted in Figures IX and X. The history of the patient was briefly as follows.

D.T., a man, aged forty-nine years, was first admitted to hospital in April, 1928, with a swelling in the middle third of the right tibia, which had been present for twelve months. His leg had been fractured on three occasions, once at the age of twelve years, again at the age of sixteen years, and for the third time at the age of thirty-six years. The first fracture was said to have been compound and spiral. After the third fracture he had no trouble with the leg for the ensuing twelve years, when the swelling complained of was first noticed. Examination disclosed a hard, lobulated enlargement of the middle of the shaft of the tibia, about 12·5 centimetres (five inches) in diameter and involving about one-fourth of the length of the bone. The skiagram (Figure IX) was reported on as follows:

> Large swelling present in middle of shaft of tibia, with thinning of cortex and expansion of bone; marked cyst formation present in expanded area . . . . appearances are consistent with those of *osteitis fibrosa*, with possible benign tumour formation added.

At operation, on May 5, the middle portion of the tibia with the tumour was excised and a dead bone graft was inserted. The tumour consisted partly of solid fibrous tissue and partly of cystic areas; and the pathologist regarded the condition as one of *osteitis fibrosa*. The graft failed, some infection of the wound occurred, and the graft was removed in October. A sinus persisted, and amputation 22·5 centimetres (nine inches) below the knee joint was performed in December, 1928.

In March, 1930, the patient returned, with some swelling in the amputation stump of several weeks' duration. A skiagram of the stump at this time (Figure X) showed a soft tissue swelling surmounting rather irregular bone ends. As the swelling continued to increase and some ulceration of the skin developed, reamputation through the lower end of the femur was performed in July, 1930. Pathological examination revealed that the bone ends were enveloped in a large mass of fibrous tissue, which infiltrated the adjacent muscles and fatty tissue. Examined microscopically, the tissue was densely fibrous and poorly cellular; but its nuclear features and infiltrative properties led to the conclusion that it was a slowly growing, lowly malignant fibrosarcoma. Subsequent skiagrams of the lungs showed no evidence of metastases, and as far as is known the patient remains well.

Review of this case suggests that the original tibial tumour must have been fibrosarcomatous, though only of low malignancy. The microscopic distinction between such a tumour and *osteitis fibrosa* would be almost impossible, and only the subsequent course of the case indicated the true nature of the growth. Attention may also be drawn to the general similarity of the skiagram (Figure IX) to that of a giant-celled tumour. Probably any lowly malignant, slowly growing central tumour of bone could produce similar expansion and skiagraphic appearances. It is also of interest to speculate regarding the possible causal relationship between the repeated fractures of the bone and the subsequent development of the tumour; but, needless to say, no definite conclusion is possible on this point.

Multiple Myelomatosis.

The only point I wish to refer to regarding multiple myelomatosis is its possible confusion skiagraphically with disseminated metastatic growths. It will be recalled that many carcinomatous metastases are predominantly osteolytic, producing well-defined spherical areas of rarefaction, as seen in skiagrams. At times it must surely be almost impossible for a distinction to be made on purely skiagraphic evidence. Figure XI, which shows skiagrams of pieces of bone obtained at autopsy from a patient with typical plasma-celled myelomatosis (see Figure XII), illustrates the radiological resemblance to metastatic carcinoma; and Cosin (1935) reports a similar case. Bence-Jones proteosuria, if present, will be a valuable diagnostic aid, but not an infallible one, since it is present on rare occasions in cases of carcinomatosis of the skeleton.

Ewing's Tumour of Bone.

In 1933 Colville and I reported a case which had been diagnosed as Ewing's sarcoma of the femur and which had exhibited perfectly the syndrome generally accepted as characteristic of that growth.

> The patient was a child. The tumour was a fusiform one affecting the entire shaft of the femur; the initial clinical and skiagraphic diagnosis was osteomyelitis. The skiagram exhibited prominent "onion-skin" laminæ (Figure II). Biopsy revealed a diffuse, round-celled, undifferentiated growth prominently subperiosteal in situation. Metastases appeared in other bones, and the early response to therapeutic irradiation was prompt and striking. On all accepted grounds, then, the clinical diagnosis of Ewing's sarcoma was thoroughly justified. Subsequent autopsy, however, disclosed a small primary adrenal neuroblastoma, with widespread skeletal metastases, of which the large femoral tumour was one.

In view of this case, and because we could discover no records of complete autopsy study in cases diagnosed as Ewing's sarcoma, Colville and I concluded that the existence of a primary bone tumour capable of producing the Ewing syndrome had not yet been established, that the syndrome probably did not denote a single pathological entity, and that metastatic growths including neuroblastoma would probably be found to be responsible in many of the cases.

Since the publication of our paper many further reports of Ewing's tumours have appeared, and the following brief review of some of these will show the unscientific confusion which still envelops the subject.

C. L. Connor, whose paper in 1926 did much to introduce the idea of Ewing's endothelial myeloma as an entity, has published a recent paper (1934), in the introduction of which he makes the following statement:

> I am now certain that among the group of tumors reported by me in 1926 as Ewing's tumor, there were a number which were undoubtedly cases of multiple myeloma or lymphosarcomatosis with metastases to bones. The usual and just criticism has been that autopsies were not performed on most of the recorded cases, and that without a complete examination *post mortem*, a primary visceral tumor could not be excluded.

Connor then proceeds to report (all too briefly) three cases of supposed Ewing's tumour, in which autopsies were performed and in which he considers the diagnosis has been proved. The first case was one of solitary tumour of the scapula, without metastases, in a man aged thirty-one years. Connor's contention regarding this tumour, that "it ought to be, by exclusion if for no other reason, an endothelial tumor of bone", cannot be accepted. The tumour responded only slightly to X ray treatment. The autopsy details are scanty, most of the organs not even being mentioned; and the cause of a general peritonitis and great distension of the stomach is left unexplained.

Connor's second case was one of tumour of the fibula in a child aged six years. There was "slight benefit" from X ray treatment. The tumour contained radiating spicules of new bone. Connor admits that the tumour resembled osteogenic sarcoma, and no information is given regarding the structure of the metastases which were present in the lungs and other bones. His third case was that of a boy, aged five years, with a tumour of the femur. The growth responded to irradiation. Autopsy disclosed growths also in the skull and lungs, the lower lobe of the right lung being entirely replaced by tumour; and the possibility that this large growth might have been a primary one is not even mentioned.

Connor's concluding remarks speak for themselves:

> From the three cases recorded here I believe we may say that the endothelioma of bone is much like the malignant endothelioma found elsewhere; that it does not respond rapidly and readily to X ray treatment but disappears gradually and slowly as compared, for instance, with a metastatic lymphosarcoma or a neuroblastoma . . . Another revision can be made with the evidence at hand. The tumor cell of Ewing's sarcoma can become an osteoblast and form bone.

Since two of the principal features of the Ewing syndrome as originally described were that the growth was highly radiosensitive and non-osteogenic, these conclusions of Connor add confusion to confusion.

Melnick (1933) has contributed a good report of the case of a girl, aged fifteen years, with a tumour of the femur, biopsy examination of which "was diagnostic of Ewing's sarcoma". (Just how the microscopic appearance of an undifferentiated round-celled growth can be "diagnostic" of anything is rather puzzling.) Autopsy revealed also a tumour at the apex of the left lung; but all other viscera, lymph-glands and bones were unaffected. No details are given, however, regarding the examination of most of the organs; and, in view of the poor response of the tumour to irradiation, and of the absence of metastases in either lymph glands or other bones (commonly present in Ewing's syndrome), the diagnosis in Melnick's case is decidedly questionable.

De Santo (1934), who agrees that many tumours are being erroneously called Ewing's tumour, nevertheless describes a tumour of the tibia in a boy, aged five years, under the title of "Ewing's tumor—primary intracortical and subperiosteal lymphangioendothelioma", a diagnosis which he reaches on histological grounds. Autopsy revealed growths in many bones, in the left lung, and a large retroperitoneal mass in the upper lumbar region. The possibility that either the pulmonary or retroperitoneal tumour may have been primary is not given the consideration it deserves.

Stewart (1933) is prepared to diagnose "endothelial myeloma" of bone from fragments obtained by aspiration. He states that:

> On three occasions we have been able by considerable search to detect the small rosettes characteristic of this tumor.

Why are rosettes "characteristic" of "endothelial myeloma", when we know them to be commonly

present in neuroblastomata? In another paper (1933) on the radiosensitivity of tumours, Stewart says of Ewing's tumour:

> Most endothelial myelomas are radio-sensitive, but occasionally one sees a resistant endothelial myeloma.

Of the radiosensitivity of neuroblastomata he says:

> The retroperitoneal and suprarenal groups are highly sensitive.

If this is so, then clearly the criterion of radiosensitivity will not permit distinction between a Ewing's tumour and metastatic neuroblastoma. This point is well substantiated also by the case reported by Colville and myself and already referred to. Incidentally, Stewart's Figure XXXII of a Ewing's tumour shows typical rosettes exactly like those seen in nueroblastomata.

Coley (1935) and Geschickter (1936) both refer to a number of cases of Ewing's sarcoma, but with neither autopsy findings nor any useful pathological information.

Porter and co-authors (1936) contribute a good account of the case of a woman, aged twenty-three years, with a tumour of the pelvis which they regard as a "Ewing's sarcoma". Autopsy revealed metastases in many bones and in the lungs and thoracic lymph glands. There were many scattered nodules in the lungs and pleura; no gross involvement of the main bronchi was present; but there were thick cords of tumour tissue parallel to the bronchi in all parts of the lungs. The adrenals contained microscopical metastases. The authors consider the possibility of the tumour being a primary carcinoma of the lung, but exclude this diagnosis because of the absence of any massive tumour and of any visible involvement of bronchial mucosa. They are apparently not aware of the existence of the infiltrating type of lung carcinoma which produces widespread peribronchial infiltration without bulky tumefaction anywhere, and the point of origin of which in the bronchial tree it may be impossible to identify. Indeed, the careful description given by Porter and co-workers of their case will enable most pathologists who are familiar with pulmonary carcinoma to make a confident diagnosis of that disease.

It is of interest to note that almost the last paper written by no less an authority than the late Carl Sternberg is a strong criticism of Ewing's tumour (1935). He concludes that not only is the conception of this tumour as a pathological entity wholly unjustified, but also that the boundaries of the clinical syndrome implied by that name are quite uncertain.

My own opinion accords completely with Sternberg's. The entire subject, in both its clinical and pathological aspects, will remain chaotic and unscientific until an adequate number of cases presenting the usually accepted syndrome have been subjected to thorough and competent *post mortem* study and the results have been properly recorded and critically evaluated.

I predict with confidence that when a number of cases have been adequately investigated it will be found that metastatic tumours are most often responsible for the so-called Ewing's tumour of bone; and indeed it will not surprise me if it is found that no such thing as a "primary endothelial myeloma" exists.

Summary.

1. Metastatic tumours in bones are very frequent; primary tumours of bones are relatively rare.

2. Metastatic tumours from symptomless or unsuspected primary growths may simulate primary bone disease clinically and skiagraphically.

3. Metastatic growths may be accompanied by the formation of new bone from expanded periosteum. This new bone may appear as radial spicules vertical to the bone surface, or as "onion-skin" laminæ parallel to the surface.

4. Osteogenic sarcomata may exhibit similar new bone formation of periosteal non-neoplastic origin. Distinction of an osteogenic sarcoma from a metastatic growth containing periosteal new bone may be difficult skiagraphically. Several points which may aid in diagnosis are discussed.

5. The so-called benign giant-cell tumour of bone is not always benign.

6. Other kinds of centrally situated non-osteogenic sarcomata of bone, for example, fibrosarcomata, may produce bone expansion and skiagraphic appearances resembling those of giant-cell tumour or of fibrocystic disease.

7. Multiple myelomatosis is sometimes imitated skiagraphically by metastatic carcinoma.

8. Review of recent reports of Ewing's sarcoma leads to the conclusion that the conception of this tumour as a pathological entity is wholly unjustified and that even the clinical syndrome implied by that name is ill-defined. The opinion is expressed that adequate study of cases so designated would show most or all of the tumours to be metastatic tumours in bones.

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THE DIAGNOSIS OF FEVERS IN SOUTH QUEENSLAND.[1]

By E. H. DERRICK.

(From the Queensland Health Department, Brisbane.)

Historical.

OUTBREAKS of fever have occurred in and around Brisbane since the earliest days of settlement. Dr. E. S. Jackson[(1)] concludes from his study of the early records that "the banes of the Moreton Bay Settlement for many years were intermittent fever, dysentery and ophthalmia". In the month of January, 1832, there were 127 cases of intermittent fever; in the whole year 1832 there were 665 cases, with four deaths.[(2)] As the total population in that year was only 983, the incidence of fever was very high. The types of fever can but be guessed at. Certainly very few, if any, of the fevers could have been typhoid, because of the low mortality.

"About 1857 Dr. Barton's books begin to show evidence of the appearance of typhoid fever in the Settlement."[(3)] In 1866 a select committee on the hospitals of the colony drew attention to the prevalence and high mortality of "colonial fever", a term more or less synonymous with typhoid fever.[(4)] Again, in 1884, a committee reported on the prevalence of typhoid fever in Brisbane and suburbs.

While typhoid fever has undoubtedly been, since its introduction, the cause of most of the mortality from fever, the other types of fever, distinguishable in general from typhoid by their milder nature and shorter duration, have contributed much to the morbidity. The incidence of these fevers has varied greatly from year to year. They have prevailed more in rural areas than in the city. Outbreaks have frequently followed the clearing of new country for settlement.

These fevers have gone by various names: "simple continued fever", "low fever", "scrub fever". They have sometimes been given locality names. Of late years the common name has been "coastal fever". Coastal fever is not an entity. It consists of at least three different diseases: leptospirosis, "Q" fever, and scrub typhus. The constituents vary in different parts of Queensland. As diagnosis becomes more accurate the term will disappear.

Classification.

The fevers met with in and around Brisbane may be classified as in Table I. The classification is quite arbitrary, but aims at being useful. Groups II and III could obviously be considerably extended.

This paper will deal mostly with the fevers of Group I. But first I shall comment briefly on some of the fevers of the other two groups.

TABLE I.
Fevers of South Queensland.

More Common Types of Fever.	Rarer Types of Fever.	Certain Fevers with a Local Focus or Non-specific Cause.
Typhoid and Paratyphoid Fevers.	Dengue Fever.	Pneumonia.
"Q" Fever.	Rat-Bite Fever.	Pyelitis.
Leptospirosis: Weil's Disease and Seven-Day Fever.	Undulant Fever.	Pulmonary Tuberculosis.
Murine Typhus.	Pyocyaneus Infection.	Septicæmia.
Influenza.	Psittacosis.	Local Infections.
	Northern Leptospirosis, Scrub Typhus, Malaria } North Queensland Fevers.	

Dengue Fever.

Dengue fever is a fever of about five days' duration, of a saddle-back type. There is an erythematous rash at the beginning and a rubeoloid rash at the end. In Queensland a diagnosis of dengue fever may be made only during an epidemic. There has been no epidemic and therefore no dengue fever in Queensland since 1926.

Rat-Bite Fever.

Rat-bite fever is rare. A search of hospital records in Brisbane revealed only one case in the last fourteen years. The diagnosis will be made by the characteristically recurring fever and the history of a rat bite.

Undulant Fever.

Undulant fever is caused by three species of *Brucella*—*Brucella melitensis*, *Brucella abortus* and *Brucella suis*. These may be termed the goat, the cow and the pig types, although there is some overlapping. The only one of the three in Queensland is the cow type, *Brucella abortus*, the least virulent of the three. This is very common in our dairy cattle and causes contagious abortion. The germ is common in the milk supply, yet undulant fever in man is quite rare here. One Queensland case, a rather doubtful one, has been reported in the

[1] A post-graduate lecture delivered in Brisbane on May 28, 1938.

literature.[5] Infection occurs rather by direct contact with infected cows, as in dairy farming, veterinary work, and perhaps in abattoir work, than by ingestion of infected milk.

The possibility of undulant fever should be kept in mind when a man engaged in one of these occupations develops an irregular fever. The laboratory test is the agglutination of *Brucella abortus* by the patient's serum.

Pyocyaneus Fever.

General infection with *Bacillus pyocyaneus* (*Pseudomonas aeruginosa*) is apt to follow ingestion of contaminated water. A minor epidemic, with nine proved cases, occurred in Townsville in 1926 and 1927.[6] The symptoms are rather like those of typhoid fever. The course is two or three weeks or longer. Leucocytosis is a feature. The diagnosis is made by culture of the organism from the blood or fæces early in the illness, or by the agglutination test later in the illness or in convalescence. I have been unable to detect an example of this fever in Brisbane.

Psittacosis.

No case of psittacosis has been detected in the course of our investigations; yet one cannot doubt that human psittacosis occurs in Queensland as in Victoria, and that cases will eventually be found if looked for. Fever patients should always be questioned as to their association with cockatoos and parrots.

In psittacosis the fever has an acute onset, lasts one to three weeks and falls by lysis. Headache is constant and intense. Cough commonly appears after the first few days. Diarrhœa is frequent in Australian cases. The patient may feel bright early in the illness, but later passes into a typhoid state. The pulse is comparatively slow. Crepitations and, later, patchy areas of consolidation may be found in the lung. Sputum is scanty.

The laboratory diagnosis is achieved by the inoculation of mice with blood or sputum from the patient, and the demonstration of the specific psittacosis bodies in their spleens four to ten days later.[7] Blood is infective only in the first three days of the illness. Sputum is often difficult to obtain, and frequently carries also pneumococci and other organisms pathogenic to mice. Thus neither the clinical nor the pathological diagnosis is easy, and the best clue is the history of contact with sick birds. The birds suspected of transmitting the illness should also be examined.

North Queensland Fevers.

Neither scrub typhus nor malaria is endemic in the southern division of Queensland, and is likely to be found here only in visitors from the tropics. Nor have the types of leptospirosis of the northern sugar fields been found in the south.

Pneumonia.

Pneumonia of the classical type, with its painful and rapid breathing, cough, viscid rusty sputum and obvious lung signs, is easy to diagnose. But a "larval" pneumonia has on several occasions caused difficulty. The diagnosis in these cases was established by radiographic examination of the chest, confirmed by the failure of the tests for the specific fevers.

Urinary Infections.

Pyelitis or pyelonephritis may cause obscure fever; and the associated rigors are often attributed to malaria by a patient who has once lived in a malarious country.

Typhoid and Paratyphoid Fevers.

Typhoid Fever.

Typhoid fever is the most serious of the local fevers, and causes the highest temperatures. It begins insidiously as a rule, with gradually rising temperature. In the second week the fever is high and continued, then becomes remittent and gradually falls. The average duration is about four to six weeks.

The clinical diagnosis is made on the gradual onset, the high fever, at first continued and later remittent, enlargement of the spleen, the absence of the superficial abdominal reflexes, and the presence of rose spots. Diarrhœa is not a common symptom, but if present will help in the diagnosis.

Figure I is readily recognizable as the temperature chart of a typhoid patient. The chart in Figure II, however, is not typical, and alone would

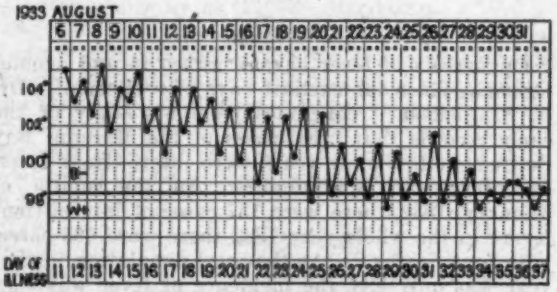

Figure I.

Typhoid fever, typical temperature chart. *W* = Widal reaction, *B* = blood culture.

hardly be accepted as indicating typhoid fever. The patient whose temperature chart is shown in Figure II stated on admission to hospital that he had been ill for two weeks; but as the serum then did not agglutinate *Eberthella typhosa*, the actual period was probably only about one week.

The laboratory diagnosis of typhoid fever may be made early in the illness, while the fever is high, by culture of *Eberthella typhosa* from the blood. The organism may also be cultured from the fæces or urine. In the second week or later the agglutination test is the method of choice.

Typhoid is the most common continued fever in females in Brisbane. The other fevers, except perhaps murine typhus, have an occupational incidence almost exclusively affecting males.

Paratyphoid Fever.

Paratyphoid fever resembles typhoid fever. The course tends to be shorter and milder and the fever more irregular. The rash is usually like that of typhoid, but the spots may be larger and more profuse. The diagnosis is made by the same clinical features as in typhoid fever, and by

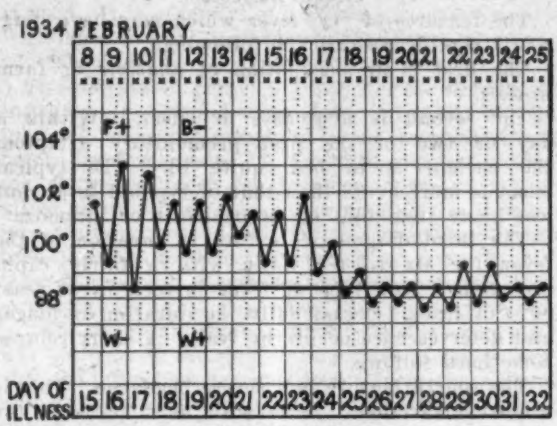

FIGURE II.

Mild typhoid fever. *F* = fæcal culture, *B* = blood culture, *W* = Widal reaction.

the same laboratory tests. Blood culture is not, however, so often successful in paratyphoid unless it is carried out quite early in the illness.

The term paratyphoid is often used loosely to designate an illness like typhoid, but which is not typhoid. When the term is confined to infection with *Salmonella paratyphi* (paratyphoid *A*) or *Salmonella schottmülleri* (paratyphoid *B*), paratyphoid fever is quite rare in Brisbane. Of the presence here of paratyphoid *C* I have not found evidence.

The relative frequency of the individual infecting agents of the enteric group in and around Brisbane is shown in Table II. The figures are drawn from two sources, records of the patients admitted to the Brisbane General Hospital during the five years from July, 1931, to June, 1936, and the records of cases diagnosed in the laboratory of the Queensland Health Department during the three years from July, 1935, to June, 1938. Overlapping of cases has been avoided. Cases diagnosed by the isolation of the bacilli are shown separately from those diagnosed by agglutination. Some lack of precision is caused by four patients, examination of whose serum showed multiple agglutination. Altogether there were 111 patients, of whom 101 or 102 had typhoid fever and 9 or 10 paratyphoid fever. On these figures typhoid fever appears to be about ten times more common in Brisbane than paratyphoid fever.

Clinical Histories.—Clinical histories of the two cases in which *Salmonella paratyphi* was isolated are appended. The illness in the first case was like a mild typhoid fever; that in the second case was of the food-poisoning type, and progressed to a fatal ending seventy-one hours after the onset of symptoms.

CASE I (by courtesy of Dr. D. A. A. Davis).—G.Q., a storekeeper, aged fifty-four years, was admitted to hospital on January 24, 1936, with a history of illness, beginning like influenza, of ten days' duration. Four days before his admission to hospital abdominal pains and vomiting had begun. There was no complaint of diarrhœa, but the bowels were well open. There was some cough, with a little sputum and a sore throat. The temperature on admission to hospital was 39·1° C. (102·4° F.) and the pulse rate was 104 beats per minute. The tongue was furred. Fine râles were audible at the bases of the lungs. The spleen was not palpable. No rash was observed. The temperature fell by lysis, reaching normal on February 4. The fever was thus of twenty-one days' duration, or fifteen days if January 20, when the gastrointestinal symptoms began, is taken as the day of onset. (See Figure III.) A leucocyte count was made on January 30. The total leucocytes numbered 7,300, of which 62% were neutrophile cells, 34% were lymphocytes, and 4% were eosinophile cells. The Widal test elicited a positive reaction with *Salmonella paratyphi* on February 3, and the bacillus itself was cultured from the fæces on February 11.

CASE II (by courtesy of Dr. E. Russell).—W.T., an engine driver, aged thirty-eight years, was admitted to hospital on October 20, 1932, with severe diarrhœa and repeated vomiting during the previous hour. There were also cramping pains in the abdomen, legs and arms. He was cold, blue and pulseless. Under treatment, including the free administration of saline solution, he improved for a while and the temperature rose (Figure IV). He died, however, on October 23.

TABLE II.

The Relative Frequency of Typhoid and Paratyphoid Fever in and around Brisbane.

Infecting Organism.	Cases in which the Infecting Organism was Revealed by Culture.			Cases in which the Infecting Organism was Revealed by the Presence of Serum Agglutinins.			All Cases.	
	Brisbane General Hospital.	Queensland Health Department.	Combined Figures.	Brisbane General Hospital.	Queensland Health Department.	Combined Figures.	Diagnosis.	Number of Patients.
Eberthella typhosa	15	11	26	21	54+1[1]	75+1[1]	Typhoid fever.	101
Salmonella paratyphi	2	0	2	0+2[1]	1+1[1]	1+3[1]	Paratyphoid fever, *A* and *B*.	9
Salmonella schottmülleri	0	0	0	3+2[1]	0+2[1]	3+4[1]	Doubtful, typhoid or paratyphoid fever.	1
Total	17	11	28	26	57	83	Total	111

[1] In the serum of these patients agglutinins were found, apparently equally significant, against two of the bacilli. It was not possible on the evidence to say which was the infecting bacillus

At the autopsy, performed by Dr. J. V. Duhig, hæmorrhagic gastritis, injection of the jejunum, and enteritis at the terminal part of the ileum were found. The spleen was much enlarged and rather soft, and from it *Salmonella paratyphi* was cultured.

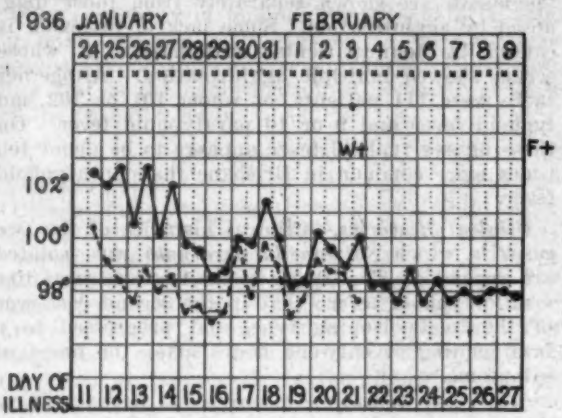

Figure III.
Paratyphoid *A* fever. *W* = Widal reaction, *F* = fæcal culture.

"Q" Fever.

"Q" fever is a Rickettsial disease which occurs both in Brisbane and in the countryside. Presumably some animals of the bush are reservoirs of

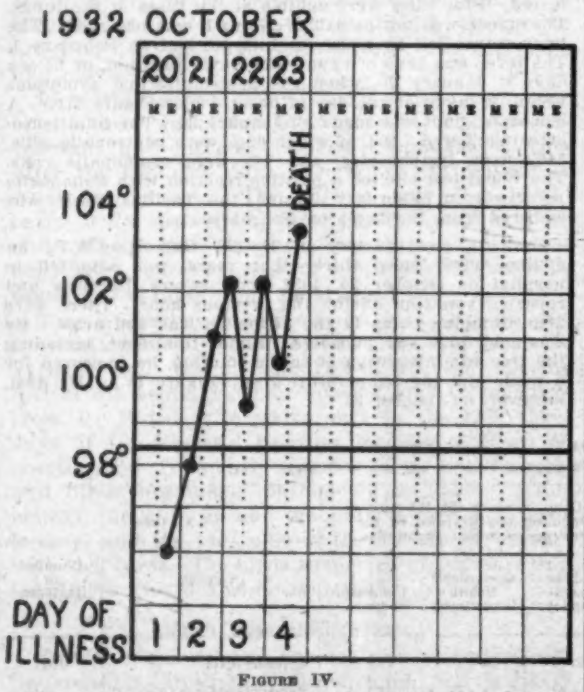

Figure IV.
Temperature chart of fatal case of paratyphoid *A* fever.

infection and some blood-sucking parasite is the vector. The country cases have occurred over the area extending from just north of Brisbane to Monto. Most of the patients have been workers on dairy farms close to scrub (rain-forest) areas.

In Brisbane the abattoir is the outstanding centre of infection. Of 42 cases occurring in Brisbane, only one has had no relation to the abattoir. Workers in any part of the abattoir are liable to infection, even those, like carpenters and mechanics, who have no direct contact with animals or with meat.

Clinical Diagnosis.

The features of "Q" fever which may have diagnostic significance are the following.

The first is an occupation of abattoir or farm worker.

The second is acuteness of onset. Within a day or two of the first premonitory symptom the patient is in bed, quite ill. The typical onset is more acute than that of typhoid fever, but less acute than that of leptospirosis or influenza.

The third diagnostic feature is duration of the fever for six to ten days, with a fairly rapid cessation. This type of fever is seen in at least 80% of cases. Occasionally the duration is longer and defervescence occurs by lysis. A short relapse sometimes follows.

The fourth feature is a severe headache. This is almost invariable. It is with most patients the chief complaint, and is often associated with insomnia.

The fifth is a comparatively slow pulse. This is not of much help in diagnosis, for it is present in other fevers also.

Photophobia is the sixth feature. This is a frequent sign, and may perhaps help in the diagnosis. The patient lies with eyelids half closed to exclude the light.

An absence of other diagnostic signs is the seventh feature. There are as a rule no rash, no enlargement of the spleen, no diarrhœa and no respiratory signs. There is no alteration in the total number of leucocytes or of polymorphonuclear leucocytes. A lymphocytosis gradually develops during the fever and becomes quite definite after the temperature falls.

Specific Diagnosis.

There are two methods in use for the laboratory diagnosis of "Q" fever. The first is inoculation of guinea-pigs with blood or urine from the patient. The blood should be taken during the febrile period. The specimen may be citrated to facilitate subsequent injection. If it is allowed to clot, the serum is separated and the clot is ground up for injection. Samples of blood may be sent from the country, as they retain their infectivity quite well for short periods.

This test depends on the fact that the guinea-pig is readily susceptible to "Q" fever, and responds with a febrile reaction. One attack makes the animal immune, and this immunity is specific.

If "Q" fever is the likely clinical diagnosis, and if the blood is obtained at the height of the fever, it is inoculated into two guinea-pigs, one new one and one rendered immune to "Q" fever by an earlier attack. A characteristic febrile reaction in the new guinea-pig and none in the immune one will establish the diagnosis of "Q" fever.

If the blood to be tested has been inoculated into one guinea-pig only, and it reacts with fever, then its blood or liver is used for the inoculation of a test pair.

The virus may be present also in the urine of human patients, but the results achieved by injection of urine are much less consistent than when blood is used. It is noteworthy that the successful results with urine are obtained late in the illness or during convalescence.

The inoculated guinea-pig may get an unapparent infection if the infecting dose is small, as, for instance, when the sample of blood is obtained just before the fever ends. If, therefore, the test guinea-pig shows no fever for fourteen days, it is then killed and a strong spleen-liver emulsion is injected into a second guinea-pig. By this procedure it has been possible to detect a number of cases of "Q" fever that would otherwise have been missed.

The guinea-pig method of diagnosis is very useful; in fact, the establishment of "Q" fever as an entity was built up on the specific immunity of the guinea-pig. But the test has one defect from the clinical aspect: it takes two to four weeks before the result can be given.

The second method of laboratory diagnosis is Burnet's agglutination test.[8] Burnet has been able to prepare a fairly pure emulsion of "Q" Rickettsiæ by heavily infecting a number of mice, grinding up their livers or spleens with saline solution to form an emulsion, centrifuging slowly to throw down the fragments of cells, and then centrifuging rapidly to throw down the Rickettsiæ. He suspends the deposit of Rickettsiæ in saline solution and centrifuges several times to purify it.

Such an emulsion is agglutinated by the serum of a "Q" fever patient, and this reaction may be used for diagnosis. As with other agglutination tests, a positive result cannot be obtained at the beginning of the illness. The blood for testing is preferably taken on or after the fourteenth day. This test may be used for retrospective diagnosis, for the blood may continue to yield a positive reaction for a year or more after the attack.

The Leptospiroses.

There are four types of leptospirosis in Queensland: (i) classical Weil's disease, due to the *Leptospira icterohæmorrhagiæ*; (ii) and (iii) the leptospiroses, often called Weil's disease, of the sugar fields of north Queensland, due to *Leptospira australis A* and *B*; (iv) the mild leptospirosis or seven-day fever of south Queensland, due to *Leptospira pomona*.

In south Queensland we are concerned only with types (i) and (iv).

Weil's Disease.

Weil's disease has a sudden onset, with shivers, headache and generalized pains. The fever is rather irregular. Typically it lasts about ten or eleven days, falling by lysis. A relapse usually occurs about the end of the third week. Intense injection of the eyes is often present and has diagnostic value. Jaundice often appears between the third and sixth days. A polymorphonuclear leucocytosis is the rule, and forms a point of distinction from catarrhal jaundice, which causes a relative lymphocytosis.

The important clinical points in the diagnosis are the sudden onset, the severe pains, the injected eyes and the jaundice. An occupation of sewer worker is suggestive. Weil's disease can occur in a mild form without jaundice. The diagnosis is then more difficult and depends largely on the laboratory tests.

Seven-Day Fever.

The common mild leptospirosis or seven-day fever of south Queensland is due to a distinct leptospira, *Leptospira pomona*. The fever lasts about a week. Jaundice never occurs, and there is no mortality rate. The leucocyte picture in the few cases studied has not been of any constant type.

The features of this disease that have diagnostic value are: first, its liability to occur in epidemics after heavy rain. For instance, at Beaudesert there was heavy rain on March 15 and March 16, 1937. Between March 23 and April 1 more than 20 patients with leptospirosis were admitted to Beaudesert Hospital.[9] The second significant feature is a hyperacute onset: the patient is usually in bed within a few hours. The third is the fact that the fever remains high for a day or two, then falls by lysis, the duration being from three to eight days. The fourth diagnostic feature is severity of the pains in back and limbs. The fifth is injection of the conjunctivæ, with soreness of the eyes.

Laboratory Diagnosis.—Laboratory diagnosis of the leptospiroses is made in the following ways.

1. Leptospiræ may be found in the patient's blood by double or triple centrifugation of citrated blood.
2. Leptospiræ may be cultured from the blood in a serum medium, such as Schüffner's medium. This is perhaps the simplest method of laboratory diagnosis. Leptospiræ are present in the blood only while the patient is febrile.
3. Guinea-pigs may be injected with blood taken during the febrile phase.
4. Leptospiræ may be seen in the centrifuged urine by dark-ground microscopic examination. Leptospiræ do not appear in the urine before about the tenth day of the illness. They may remain there for weeks or months.
5. Urine may be injected into a guinea-pig.
6. Agglutination tests may be made. Agglutinins begin to develop in the serum about the sixth day. Tests for their presence may be made at any time thereafter in the illness, or during convalescence. The agglutinins may persist in the serum for years. As there is only slight cross-agglutination, if any, between the various types of leptospira, it is necessary to test the serum against a culture of each local type. That means two (*Leptospira icterohæmorrhagiæ* and *Leptospira pomona*) for south Queensland, or four if the two northern types are included.

Murine or Endemic Typhus.

Murine or endemic typhus is a disease of rats, caused by the *Rickettsia mooseri*, spread from rat to rat and occasionally from rat to man by the rat flea, *Xenopsylla cheopis*.

It is present in cities, especially coastal cities, throughout the warmer parts of the world. Every year a few cases occur in Brisbane, Ipswich, Gympie, Rockhampton and probably other Queensland cities. There were over 200 cases around Toowoomba in 1926, at the same time as a plague of mice occurred.

The scrub typhus of north Queensland is a different disease, although the course is rather similar.

The features of murine typhus that point to the diagnosis are the following. The first is an occupation that brings the patient into contact with rats. Most patients in Brisbane come from the produce stores and markets of Roma Street. The second is an acute onset, more acute than with typhoid fever. The third diagnostic feature is a fever that lasts about fourteen days and falls by rapid lysis or crisis. Figure V illustrates a typical course, except for the intermission on the tenth day. Figure VI illustrates an atypical course, in which the fever fell by gradual lysis. The fourth feature is a severe headache. The fifth is a papular or macular rash, appearing roughly from the fifth to the seventh day, first on the abdomen and chest, then on the back and limbs. It is much more extensive than the rose-coloured spots of typhoid fever. It lasts about two days. It may be quite faint, but when it is pronounced it makes the diagnosis definite. The sixth diagnostic feature is a positive reaction to the Weil-Felix test. The serum of a patient with murine typhus agglutinates *Proteus X* 19. A non-motile or *O* strain of the bacillus, *Proteus OX* 19, is used for the test. This test finds its value in the second week of the illness.

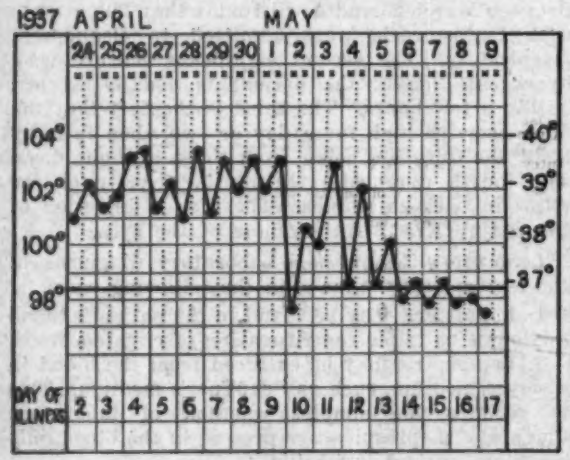

Figure V.

Murine typhus. This temperature chart is typical, except for the intermission on the tenth day.

Murine typhus is quite like "Q" fever in the early stages, which are characterized by an acute onset of a high fever with severe headache. The resemblance continues until the rash comes out, and the Weil-Felix test can be utilised to distinguish it.

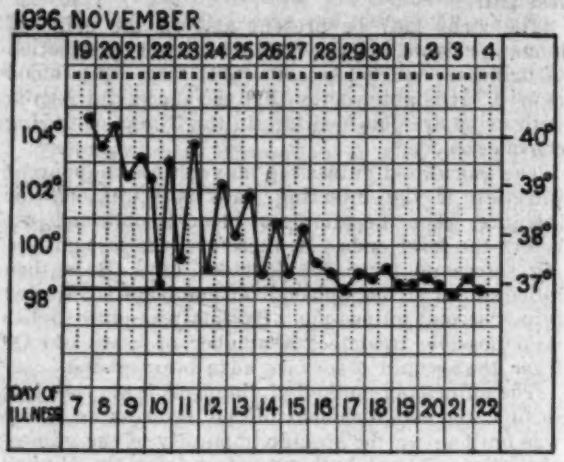

Figure VI.

Murine typhus, atypical course. Defervescence by gradual lysis.

Influenza.

The diagnosis of influenza is often made rather loosely. Many cases of leptospirosis are labelled influenza; and many other cases of leptospirosis and "Q" fever have been labelled influenza at the beginning of the attack. When the patient has still been ill after four or five days the diagnosis has needed revision.

Influenza occurs typically in epidemics, and is accompanied usually by catarrhal symptoms—coryza, sore throat, cough. But it also occurs without these symptoms; and there will then be great difficulty in distinguishing clinically between influenza and the abortive forms of "Q" fever and mild leptospirosis.

Scheme of Diagnosis.

I have collected into a scheme some of the points on which a diagnosis may be based. In typical cases of these fevers the diagnosis will be comparatively easy. In atypical cases, especially the mild abortive types, the diagnosis will naturally be more difficult. But if a careful clinical study of the patient is made and the full resources of the laboratory are utilized, a fair degree of precision in diagnosis will be attained.

When a patient with fever confronts the clinician, the following investigations will be found helpful in diagnosis.

Occupation:

Abattoir workers are likely to have "Q" fever, seven-day fever, undulant fever.
Farm workers are likely to have "Q" fever, seven-day fever, undulant fever.
Produce merchants, marketers *et cetera* (rat contact) are likely to have murine typhus fever.
Sewer workers are likely to have Weil's disease.
(All the above fevers occur mostly in males.)
Contact with cockatoos and parrots suggests psittacosis.

Mode of Onset:

A gradual onset, the patient taking nearly a week to take to bed, suggests typhoid fever.
An acute onset (one or two days) suggests typhus fever, "Q" fever.
A very acute onset suggests leptospirosis, influenza.

Type of Fever:

High and continued elevation of temperature suggests typhoid fever, typhus fever, "Q" fever, psittacosis.
Elevation lasting more than two weeks suggests typhoid fever, "Q" fever (exceptionally).
Elevation lasting about two weeks, with rapid fall, suggests typhus fever.
Elevation lasting six to ten days, with rapid fall, suggests "Q" fever.
Elevation of temperature for one or two days, then fall by lysis, suggests seven-day fever, influenza.
Indefinite temperature suggests undulant fever, variant and mild cases of other fevers.

Symptoms and Signs of Diagnostic Significance:

A rash (rose spots) suggests typhoid fever, paratyphoid fever.
A rash, papular or macular, suggests typhus fever.
A rash, erythematous at first, rubeoloid later, suggests dengue fever.
An enlarged spleen suggests typhoid fever, paratyphoid fever.
Respiratory symptoms suggest influenza, pneumonia, typhoid fever, psittacosis.
Absence of superficial abdominal reflexes suggests typhoid fever, paratyphoid fever.
Diarrhœa suggests typhoid or paratyphoid fever.
Pyuria suggests pyelitis.
Severe headache suggests typhoid, typhus or "Q" fever or psittacosis.
Generalized pains suggest leptospirosis or influenza.
Congested conjunctivæ suggest leptospirosis.
Jaundice suggests Weil's disease; it rarely means typhoid, typhus or "Q" fever.

Blood Culture:

A blood culture should be attempted when typhoid fever, paratyphoid fever, septicæmia, pyocyaneus fever or leptospirosis is suspected. Leptospiræ require a serum medium such as Schüffner's. Attempts at blood culture are most likely to be successful early in the illness, while the fever is high. As antibodies develop in the serum the chance of success diminishes.

Guinea-Pig Inoculation:

A guinea-pig should be inoculated with blood taken while the fever is high. This will decide the diagnosis of "Q" fever, undulant fever, rat-bite fever and psittacosis. Mice are better than guinea-pigs for rat-bite fever and psittacosis.

Leucocyte Count:

Leucocytosis suggests pyocyaneus infection, pneumonia or Weil's disease.
No obvious change in the leucocyte count suggests typhus fever or "Q" fever. Lymphocytosis follows later.
Leucopenia suggests typhoid fever, malaria, influenza or dengue fever.

Agglutination Tests:

Serum should be sent for agglutination tests after the first week. A positive result cannot be expected in the first five days of illness. In the second week and later the test becomes of great value. A rising titre of agglutination is more significant than the result of a single test. Previous typhoid-paratyphoid vaccination may be the cause of an agglutination.

The following diseases may be diagnosed by agglutination tests: typhoid and paratyphoid fever, typhus fever (*Proteus OX* 19 being used for murine and *Proteus OXK* for scrub typhus), a pyocyaneus infection, undulant fever, leptospirosis or "Q" fever.

Other Tests:

Culture of fæces may reveal typhoid or paratyphoid fever.
X ray examination of the chest may confirm the presence of mild pneumonia.
Dark-ground microscopic examination of urine for leptospiræ should be made.
Guinea-pig inoculation with urine may reveal leptospirosis or "Q" fever.
Culture of urine is useful in the diagnosis of pyelitis and typhoid or paratyphoid fever.
Injection of sputum into mice may reveal psittacosis.
Examination of cerebro-spinal fluid is helpful.

Acknowledgements.

I am indebted to Sir Raphael Cilento, Director-General of Health and Medical Services, Queensland, for permission to publish this article. To Dr. Alex Murphy I am grateful for advice on certain clinical points. The figures showing the incidence of typhoid and paratyphoid fevers at the Brisbane General Hospital are given by the courtesy of Dr. A. D. D. Pye, Medical Superintendent, and the notes and charts of fever patients by courtesy of Dr. E. Russell, Dr. A. Murphy, Dr. D. A. A. Davis, Professor J. V. Duhig and Dr. L. W. Gall.

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ARTIFICIAL CRANIAL DEFORMATION IN NEW BRITAIN.

By Edward Ford.

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The custom of artificial deformation of the head has come down from ancient times, and is still practised in many centres. Traces of it are widespread, and it has been recorded in scattered areas in Europe, Africa, Western Asia, Malaya, Indonesia, Melanesia, Polynesia and in South America and North America. Of all the continents Australia alone provides no evidences of its existence. For although it was undertaken in the Torres Strait and in the islands to the northward, no traces have been found on the Australian mainland.

The custom existed in ancient Egypt as early as the eighteenth dynasty (*circa* 1370 B.C.), and Professor Elliot Smith suggested that, together with certain other Egyptian practices, it spread thence round the world.[1]

Dawson offers a suggestion as to the origin of the practice in Egypt at this time.[2] Amenophis IV (also known as Akhenaten), the ruler of the period, is portrayed in contemporary sculptures as possessing a peculiarly elongated head, and his daughters are similarly depicted. This was considered to be an artistic convention of the time until Elliot Smith, in 1912, showed the head of Amenophis to be hydrocephalic, and later suggested that the heads of his daughters may have been artificially deformed. Evidence in support of this theory was supplied in 1918 by Sobhy, who showed the head of a female mummy of this period which was undoubtedly so deformed.[3] Dawson suggests that the pathologically misshapen head of Amenophis may have been copied artificially in his daughters, and that the fashion, so started, thereafter became perpetuated.

Apart from the various types of unintentional cranial deformity, which may be caused, for instance, by the pressure of head coverings or cradle boards during early infancy, the intentional deformations practised vary widely in the nature and degree of distortion displayed. Six main methods have been used in their production: by moulding of the skull after birth; by the application of boards, bandages, pads or stones; and by the pressure of cradle boards. Complex and for the most part unsatisfactory classifications of deformed crania have been drawn up from time to time, though Dingwall, in his comprehensive study of the subject, rejects these for the simpler method of grouping according to the apparatus used in producing the deformity.[4]

The Arawe Head Deformation.

The custom is practised in the Arawe district of New Britain and is frequently termed the Arawe deformation. The area in which it occurs extends along the south coast of the island of New Britain for a distance of about 300 miles, commencing at Yumbon village, towards the western end of the island, and extending eastward to and including part of the Mokolkol area. It also occurs on the adjacent islands, including Arawe (Liebliche), Kaurumutei, Makro, Bililo, Alelep (Passis Manua), and Milengloe. The practice does not extend right across the narrow island to its northern coastline, although, as Parkinson noted, there was much friendly intercourse with the natives of that part.[5]

In this area deformation is performed upon male and female infants alike, and with a few exceptions is applied to all persons in the communities. The resulting effect is the production of a conical or cylindrical elongation of the skull, from which these natives are frequently termed "long-heads" or, by German writers, *"Spitzköpfe"*.

Though the procedure varies somewhat in its detail from village to village throughout the Arawe area, the method described in the following account, mainly from the observations of Mr. H. E. Woodman, generally applies:[6]

> After the birth of an infant the mother rests for about a week. She then bathes and replaces the fibre skirt, which was worn during her period of confinement, with a new one. The infant is then washed, and a mixture of wood ashes and water prepared. With this the child's head is anointed and the mother, using both hands, massages and squeezes the crown of the head upwards. A length of bark cloth (*mal*) is then bound spirally round the head, with firm pressure, and fastened in position by a length of native string. In this way an even encircling pressure is applied to the cranium. In front the binding extends distally for a variable distance, sometimes covering the supraorbital ridges, and posteriorly the pressure is made over the occipital region. The bandage is kept in position until next morning, when it is removed. Then the massage and painting are repeated and the binding reapplied.

This process is repeated each day for about two months. The natives state that by this time the head is "set"; and though bindings are worn after this period, they are used rather as a hat or a support than for the purpose of shaping the head.

If the child cries frequently the binding may be removed for a period of about eight hours or so, and is then replaced. Chinnery[7] states that frequent crying, with the consequent abandonment of the process, accounts for the presence of a certain number of persons with normally shaped heads among those people who practise the custom, although, as will be shown, there are also other reasons for this. It was noted by Kane that, although children usually did not cry when the bandages were in position, they sometimes did so when these were loosened, and the crying continued until the pressure was reapplied.[8]

In the early stages of the treatment the great supraorbital pressure causes the eyeballs to protrude considerably, and the infant sleeps with the eyelids separated. This latter often persists into adult life, and the Reverend G. H. D. Voss has noted that these natives nearly always sleep with the eyes half open. At the commencement of sleep the eyes are quite closed, but after a time the eyelids move apart until they are almost half open.[9]

Though no reasons are given for the custom, the deformity is considered to be a character of beauty, and men with deformed heads are more desirable in the eyes of the women. Dingwall points out that in one respect at least cranial distortion differs from most other mutilations, since, as far as can be seen at present, it has no connexion whatever with initiation ceremonies or with rites occurring at puberty.[4]

As previously mentioned, it is usual to find, in villages where the practice generally applies, a number of persons who show no deformation of the head. This applies also to villages in the hinterland where there is little outside contact, and was noted by Parkinson[5] before European influence had left its mark upon the natives of the area. Although

these untreated individuals are not despised, as Chinnery[7] noted, yet they are regarded as unfortunate, and are sometimes teased by their fellows. The reasons for their condition are varied. As already mentioned, the relinquishment of bandaging on account of the continued crying of the child, accounts for only a minor number. Up to the present time the main part could be ascribed to laziness on the part of the mother, the ne'er-do-wells of the village tending to neglect the care of their children in this as in other matters. The degree to which the process was carried previously also depended upon the assiduity of the parents, careful and attentive mothers producing more pronounced deformation than careless ones. Of late years the number of unbound infants has gradually increased, and may be expected to do so further, as the result of intercourse with peoples who do not practise head deformation. The younger natives leave their districts as indentured labourers, and on return some of them do not bind the heads of their children.[8] The effect of this absence from their people is probably heightened by the fact that they are frequently derided by undeformed natives on account of the shape of their heads. The result of communication with outsiders is further shown in that the further one goes into the bush, the greater is found to be the degree of head deformation among the villagers. The bush people have their heads almost shaved, the better to display the elongation, while the coastal natives, in some touch with civilization, tend to be ashamed of it and wear the hair long and well fuzzed to hide the deformity.[9]

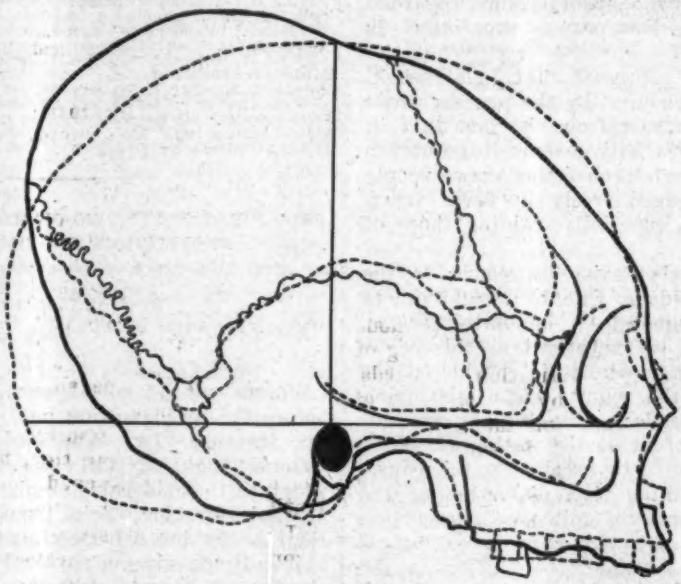

Figure I.

Superimposed outlines of an artificially deformed and a normal New Britain skull. The distorted skull is represented by a continuous line, the normal by a broken line. (Half natural size.)

The Effects of the Cranial Deformation.

Great difference of opinion exists in regard to the effect of cranial deformation upon the mental state of those so treated. This applies to the Arawe natives, as to other peoples who have practised the custom in one or other of its forms.

This conflict of opinion has probably arisen mainly from the recording of casual observations, and also from the tendency to make comparisons, not with undeformed individuals of the same community, but with others who are perhaps of different race and mental standard. For this reason characters common alike to both deformed and undeformed members of a community have been held to be the result of cranial distortion.

The opinion is widely held in the Mandated Territory of New Guinea that, owing to the practice of the custom, the Arawe natives are lacking in intelligence. This view is reflected in the literature upon the subject. Cilento stated that "Personal observation of a few cases has led the writer to the conclusion that the power of concentration, attention, and sense of responsibility are deficient in natives so treated . . .", although he acknowledged that his observations on these points were too meagre and haphazard to justify a general deduction.[10]

The opinions of several responsible persons who have lived among these people were sought in regard to this view. Among these may be especially mentioned Mr. H. E. Woodman, formerly assistant district officer at Gasmata, the Reverend G. H. D. Voss, of the Melanesian Mission, Gasmata, and the Reverend Harold Thompson, of Arawe. The latter two gentlemen, in addition to living among the people, have also conducted schools among them. The opinions expressed were based upon the comparison provided by the presence of undeformed persons in the villages. Excepting in regard to one detail, which will be mentioned later, these observers all agreed that the practice of cranial deformation caused no obvious effect upon the mind, and that in this regard deformed persons did not greatly differ in any respect from comparable undeformed ones. There is general agreement in that there is no observable effect ascribable to the practice upon general intelligence or upon concentration, attention or sense of responsibility. This statement is in agreement with that of Reche, who considered that mental capability did not appear to be the least affected by the deformation, and that individuals with deformed heads made efficient labourers.[11]

Although agreeing in the main with the general opinion expressed above, one observer considered deformed individuals to be slightly slower in response than others, and to display less rapidity

of movement, although in school work no obvious difference was observable between the two groups.[9]

The opinion was also generally held that much of the attribution of stupidity to the Arawe people was based upon their unwillingness to learn new fashions, but that this was a characteristic of the people generally, whether deformed or not. They are the most primitive on the island of New Britain, and have retained more than any others their native customs. Among these may be mentioned the immolation of widows by strangulation, which, though suppressed in the coastal districts, is said to be still practised in the hinterland. There is a tendency to compare them with other tribes whose mode of life has long since been altered by contact with civilization, and who are therefore regarded more favourably by Europeans, especially as servants or labourers.

The view sometimes expressed that an abnormal degree of docility is produced by the process needs little comment. For, apart from the fact that in this respect the deformed native varies in no noticeable degree from the undeformed, the Arawe people have in the past engaged freely in fierce tribal fighting, the islanders, especially, raiding those of the mainland.

Contrary opinions also exist in regard to the mortality of the procedure, Cilento[10] and others stating that not infrequently it has fatal results. Careful inquiries fail to substantiate this view, however, for the infant mortality is considered not to be higher than in the comparable neighbouring villages, where the heads of the infants are not bound. Further, no infant death was known which could be directly or indirectly ascribed to this cause.

It has also been stated that distortion of the skull is a potential cause of epilepsy in later life, and inquiries were made in this regard. No figures are obtainable of the frequency of epilepsy in the Arawe area; but it is considered that, although widely isolated cases are known of deformed natives who are subject to fits, yet these are not more numerous than among natives generally who do not practise cranial deformation. It is true that this opinion is not based upon a numerical foundation; but if the skull distortion acted as stated to any great extent, a more obvious incidence of epilepsy could be expected among these people. It has been noted, in very rare instances, that absentees have been reported during census-taking on account of fits, but not more frequently here than in other districts.

Summary.

The practice of artificial cranial deformation among the natives of the Arawe district of New Britain is described.

Despite contrary opinions, the deformity produced has little effect upon the mind.

No pronounced infantile mortality can be ascribed to the custom.

As far as can be ascertained the cranial distortion is not productive of epilepsy, as has been recorded.

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My thanks are due to Mr. H. E. Woodman, D.S.O., the Reverend Harold Thompson, and the Reverend G. H. D. Voss, for their valuable assistance, and also to Captain G. McLaren and Mr. F. H. Taylor for the photographs.

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THE FIRST FIFTY-TWO NAMES IN THE MEDICAL REGISTER OF SOUTH AUSTRALIA.

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There were medical men in South Australia before the proclamation on December 28, 1836, as, for instance, Dr. Woodforde, who accompanied Colonel Light. With the establishment of the province a considerable number of medical men soon arrived, some for official purposes, some to practise their profession privately, and some with the main object of engaging in pastoral or other pursuits. It thus soon became necessary to have an official record of medical men practising their profession, with their qualifications. For this purpose Ordinance number 17 of 1844, "To define the qualifications of medical practitioners in this Province for certain purposes", was proclaimed. In 1880 an Act to amend the ordinance was passed, and in 1889 an Act to amend the *Medical Act* of 1880. In 1919 a new Act, "To make better provision for the Registration of Medical Practitioners", was passed; it is still the law. In 1930 the Medical Board published officially a list of all those persons who, up to November, 1919, had been registered as medical practitioners in South Australia. The names were arranged alphabetically, and the number of each registration was given; the total up to this period was 1,023. This list forms a valuable source of information.

Through the courtesy of Mr. H. T. Young, Secretary of the Medical Board, I have been supplied with a list of the full names and the qualifications, as entered in the register, of the first 52 practitioners whose names appear therein; this number takes us to the end of 1849, a period of thirteen

years. The first medical board consisted of J. G. Nash, president; William Wyatt, secretary; Handasyde Duncan, B. A. Kent and John Woodforde. At the first meeting the minutes state "Present all members of the Board", but do not enumerate them. Dr. Wyatt was secretary, and on looking at the entries in the register and as reproduced later in this article, it will be seen that the qualifications were not always described quite correctly. Thus The Royal College of Surgeons of England is often referred to as of London; there is an M.D. of France; Jena is spelt Gena; and in some cases the dates are omitted.

A list of this kind makes a good jumping-off place for information as to the medical profession in a State such as South Australia. It would be of considerable interest if similarly in the other States some account was made available of the first names on their medical registers. In South Australia nearly six years passed before an ordinance covering registration was proclaimed. During that period various medical men were in South Australia and died, or left it. Other medical men coming within the period considered in this paper who were engaged in pastoral and similar pursuits did not consider it worth while to become registered. There are, therefore, a considerable number of names to be added to the 52 here considered if the medical practitioners in South Australia before the year 1850 are to be given in full.

In the following paper much information of value has been obtained through the State Archives, Adelaide, whose archivist, Mr. G. H. Pitt, has given me much valuable information and assisted me materially. In their records they have the late Dr. A. A. Lendon's papers on medical men in South Australia, and they also have a card index of prominent South Australians referred to in *The South Australian Register*, *The Adelaide Observer*, *The Advertiser*, and other daily papers, as well as those mentioned in published works. This I have found of much value, and I am much indebted to Mr. Pitt, to Mr. Young, of the Medical Board, and to Mr. J. D. Somerville and others, for the material assistance they have rendered me.

In this account, where a biography appears elsewhere, a reference is given to it, but only a few epitomized details are included here. The object of the paper has been to indicate more particularly where sources of information are available. At the end of the paper I have given similarly some account of medical men of this period who do not appear on the register. Doubtless there are others that I have overlooked, and I shall be glad to receive information about these, as well as additional data in connexion with those who do appear on the register. Some men, such as number 63, David Wark (1807-1862), who came out as early as 1838, did not register until a later period.

1. James George Nash (1805-1880).—The name of James George Nash was registered on December 28, 1844, M.R.C.S. (London, 1827). On his arrival in South Australia by the ship *Claxton* in 1838, he "was appointed Colonial Surgeon, a position he held for some years, until he was compelled to resign, owing to impaired health, soon after which he left the colony". He died at Woodville, New Ferry Park, Cheshire, on November 12, 1880, at the age of seventy-five years. He had for some time been gradually going blind, and had suffered from an accident in falling down stairs. He was considered a very clever surgeon and was greatly esteemed. (*The Adelaide Observer*, January 8, 1881, page 78.) He was the first president of the Medical Board, holding this position for nearly twenty years. J. C. Hawker gives the following pleasant account of a gathering at Dr. Nash's house in the days of Governor Gawler (October, 1838, to May, 1841):

> There were occasionally pleasant little socials of what were considered the elite of Adelaide, and small dances were given. The residence of the Colonial Surgeon, Dr. Nash, was in a little garden on the west [*sic*] side of Grenfell Street and nearly at the east corner of Hindmarsh Square. The house was partly brick and partly wood, and the rooms were small. However, by knocking two rooms into one a fair space for dancing was obtained, and Mrs. Nash issued invitations for a fancy-dress ball. This was the first of the kind attempted in Adelaide, and for many days both ladies and gentlemen had to tax their brains to decide what they should wear. The party was a complete success. Mr. Hall and myself were amongst the invited.

J. C. Hawker was induced to stay late; but in returning to Government House in the dark, fell into the excavations being made for the cellar of the new house, in which, up to ankles in mud, he had to stand till daylight. Governor Gawler did not approve of dances and asked him not to go again. (J. C. Hawker, "Early Experiences in South Australia", 1899, pages 12 and 13.)

2. William Wyatt (about 1805-1886).—The name of William Wyatt was registered on December 28, 1844, L.W.C.A. (London, 1827), M.R.C.S. (England, 1828). William Wyatt arrived as surgeon to the *John Renwick* in the middle of February, 1837. On August 1 he was appointed *ad interim* Protector of Aborigines. He died, aged eighty-one, on June 10, 1886. A biography of him by the late Dr. A. A. Lendon, under the title "The Early Protectors of the Aborigines", is in the State Archives, Adelaide. His sound remarks as coroner in rebutting Press comments are referred to under "31. John Knott".

3. Handasyde Duncan (1811-1878).—The name of Handasyde Duncan was registered on December 28, 1844, L.R.C.S. (Edinburgh, 1829), M.D. (Glasgow, 1831), M.F.P. and S. (Glasgow, 1836). He arrived in South Australia in 1839. He resided at Port Adelaide, where he was port health officer. A biography by the late Dr. A. A. Lendon, with portrait, is in the State Archives, Adelaide.

4. Benjamin Arden Kent (about 1810-1864).—The name of Benjamin Arden Kent was registered on December 28, 1844, M.D. (Edinburgh, 1831). He is believed to have been born at Walsall, Staffordshire. He was probably at least thirty years of age, and was married and had a family when he arrived by the *Warrior* on April 17, 1840. Dr. A. A. Lendon, under the title "Kent *v.* Torrens", gives a full biographical account of him and of his lawsuit over land matters with Lieutenant-Colonel Robert Torrens, Chairman of the South Australian Colonization Commissioners, in the *Royal Geographical Society of Australia, South Australian Branch, Proceedings*, Volume XXVII, 1915-1916, page 29, and Volume XXX, 1928-1929, page 74. The portrait given as that of Dr. Kent in Volume XXVII is of some other person; this error is pointed out in Volume XXX, where the proper portrait is reproduced. He was an original member of the South Australian Medical Board, created by ordinance in 1844, and he resigned in 1850. He was in England in 1859 and died at Ryde, in the Isle of Wight, in 1864. Kent Town, a suburb of Adelaide, is named after him, and a reproduction of a sketch by Dr. A. C. Kelly of his house in what afterwards became Kent Town, appears in THE MEDICAL JOURNAL OF AUSTRALIA, April 28, 1934.

5. John Woodforde (1806[1]-1866).—The name of John Woodforde was registered on December 28, 1844, L.W.C.A. (London, 1832), M.R.C.S. (England, 1833). He was surgeon of the *Rapid*, in which Colonel Light came out, arriving at Kangaroo Island on August 19, 1836. After George Stevenson's death, in October, 1856, he succeeded him as coroner, which office he held till his death on April 11, 1866, in his sixtieth year. A biography by the late Dr. A. A. Lendon is in the State Archives, Adelaide.

6. William Durie.—The name of William Durie was registered on December 28, 1844, L.R.C.S. (Edinburgh, 1829). Certificate of qualification as assistant surgeon, R.N. (1834). No record in the State Archives, Adelaide.

7. Charles Davies (1814[2]-1888).—The name of Charles Davies was registered on December 28, 1844, M.D. (France, 1838). He arrived, at the age of twenty-five, by the *Branken Moor*, on February 5, 1840. After practising in North Adelaide, he retired to Beaumont, where he lived from 1862 to 1888, dying of heart disease on February 12, 1888, aged seventy-four years. A biography by the late Dr. A. A. Lendon is in the State Archives, Adelaide. (See also "Pastoral Pioneers of South Australia", Volume II, 1927, page 10, with portrait.)

8. George Mayo (1807-1894).—The name of George Mayo was registered on December 28, 1844, L.W.C.A. (London, 1828), M.R.C.S. (England, 1829). Later the registration of F.R.C.S. (England, 1851) was added. He came to South Australia as surgeon on the *Lady Emma*, which arrived on December 4, 1837. There is a biography by the late Dr. A. A. Lendon in the State Archives, Adelaide.

9. William James Browne (about 1815-1894).—The name of William James Browne was registered on January 4, 1845, M.R.C.S. (Edinburgh, 1838). William James and his brother Dr. John Harris Browne were the sons of a Wiltshire land-owner. Neither practised his profession to any extent in South Australia, but engaged in extensive pastoral pursuits. At the age of twenty-three, William James sailed for South Australia in the *Buckinghamshire*, in December, 1838, the voyage taking six months. The brothers were for some time in partnership and at one period were the largest exporters of wool in the colony. They owned such famous estates as Moorak in the south-east, Booborowie and Buckland Park, as well as many other properties in various parts of the colony, in some instances in partnership with others. In 1866 William James took up his residence at Moorak. He was a member of the second Parliament (1860-1862), representing Flinders in the House of Assembly. The writ of Parliament House certifying his election calls him "William John", evidently a mistake, as shown by his name in the medical register. After taking steps to open up the Northern Territory cattle runs of Springvale, Delamere Downs and Newcastle Waters, he left South Australia for good in 1878. In 1880 he contested unsuccessfully a seat in the House of Commons. He died at Eastbourne on December 4, 1894, in his eightieth year. He claimed to be the first to cross pure old Lincoln rams with the "largest fleeced and largest merino ewes". A full biographical account, with a portrait, appears in Rodney Cockburn's "Pastoral Pioneers of South Australia", Volume I, 1925, page 30, reprinted from *The Adelaide Stock and Station Journal*. (See also *The Adelaide Observer*, December 8, 1894, page 30, and May 4, 1895, page 30.)

10. John Harris Browne (1817-1904).—The name of John Harris Browne was registered on January 4, 1845, M.R.C.S. (Edinburgh, 1839). Born on April 22, 1817, John Harris joined his elder brother, William James, in South Australia, in 1840, and engaged with him in pastoral pursuits. He lived for eight years at Buckland Park, which was acquired by the brothers in 1856. In 1862 he fitted out a party under Josiah Bonnin to inspect country in the Gawler Ranges; but the report was unfavourable. Shortly after dissolving partnership with his brother he returned to England, but paid several later visits to South Australia, his last public appearance in Adelaide being at the funeral of the explorers Wells and Jones. He died in England in January, 1904, in his eighty-seventh year. John Harris Browne will be particularly remembered as the medical companion of Captain Charles Sturt in his memorable expedition of 1844-1845 into the interior of Australia. During this journey he suffered severely from scurvy. When his leader lay almost helpless and exhausted, Dr. Browne made a strenuous journey from the Depot Creek to Flood's Creek, finding water still there, but "black as ink". Thither the party quickly retreated just in time. His cheerful nature, professional skill and devoted care, as Cockburn points out, undoubtedly enabled the great explorer to survive the return journey. A biographical account, with a portrait, follows that of his brother in "Pastoral Pioneers of South Australia", Volume I, 1925, page 32; also Volume II, page 136.

11. William Innes.—William Innes's name was registered on January 11, 1845, M.R.C.S. (England, 1836). No record appears in the State Archives, Adelaide.

12. Alexander Charles Kelly.—The name of Alexander Charles Kelly was registered on January 11, 1845, M.R.C.S. (Edinburgh, 1831), M.D. (Edinburgh, 1832). He was the son of John Kelly, manager of the British Linen Company's Bank, Dunbar, Scotland, and was born in June, 1811. For about eight years after graduation he acted as surgeon in the East India Company's ships trading to India and China. He arrived in Adelaide by the *Baboo* on May 10, 1840, and was appointed an assistant surgeon at the Adelaide Hospital. Returning to the Old Country, Kelly lectured on "Australia as a Field for Emigration", but came back to Australia in 1843. He purchased an eighty-acre section of land between Morphett Vale and the sea. During his stay in Europe he had visited France and other wine-producing countries, and the South Australian climate and soil so impressed him as to their suitability for vine-growing that he determined to plant a vineyard. He communicated with Sir William Macarthur, of Sydney, and obtained a supply of vine cuttings of different varieties, and planted about thirty acres. To this vineyard he gave the name of "Trinity". The name arose in this way. Competent workmen were at this period difficult to obtain, especially outside the city; in order to obtain help to build his homestead, Kelly was compelled to secure the services of three hatters (hence "Trinity") who had come to Adelaide and had found no opening for their particular occupation. Acting under his instructions, they burnt the lime, made bricks, and sawed timber from trees on the property, and with the help of a mason and Kelly's liking for carpentry, a very comfortable house was built. One account states that he visited Scotland in 1846 as emigration lecturer, returning by the *Lady McNaughton* in the following year. He then settled down at Trinity to vine-growing and wine-making, like his neighbour John Reynell, the types of wines produced being burgundy, port and constantia. In 1859 he visited New South Wales to inquire into the methods of vine-growing there, and to obtain particulars in connexion with his work "The Vine in Australia". In 1861 he purchased some heavily timbered Crown lands on the slopes a few miles north-east of McLaren Vale, and there planted the later famous Tintara vineyard. In 1871 he went to England to open up a market for the Tintara wines, and appointed P. B. Burgoyne, with cellars in Old Bond Street, agent for the Tintara Vineyard Company. This was the commencement of Burgoyne's interest in South Australian wines. The sales of wine in London being insufficient to cover the heavy expenses of advertising *et cetera*, the company went into liquidation in 1876. Kelly then retired to Norwood, where he died on October 9, 1877. In 1855 he was married at Morphett Vale to Ann, eldest daughter of George Worthington, stipendiary magistrate, of Morphett Vale, formerly of Liverpool. ("The Cyclopædia of South Australia", Volume II, 1909, page 835; *The Standard*, Adelaide, Volume I, Number 38, July 17, 1914.)

[1] There is some doubt about the date of Woodforde's birth.
[2] There is some doubt about the date of Davies's birth.

13. Martinus Peter Hayward.—The name of Martinus Peter Hayward was registered on January 11, 1845, M.R.C.S. (England, 1839). He was surgeon in the barque *Phoebe* (Captain Dale), London to Adelaide, December, 1846. ("Reminiscences of J. F. Hayward", *Royal Geographical Society of Australasia, South Australian Branch, Proceedings*, Volume XXIX, 1927-1928, page 80.) December, 1846, is evidently a mistake, as his name was registered in South Australia in January, 1845. According to "Pastoral Pioneers in South Australia", Volume I, page 108, Martinus Peter Hayward, a brother of J. F. Hayward, at one time owned a brewery at Clare and had an interest in the Canowie run near Hallett.

14. Charles Knight.—The name of Charles Knight was registered on January 14, 1845, M.R.C.S. (England, 1840). In the State Archives, Adelaide, are two letters from Knight addressed to the then Governor, Sir George Grey, and a copy of the reply to the second one, written by the Governor's secretary. The first is dated July 30, 1841, from Fordham's Hotel, asking for an advance of £25, to purchase bedding and other necessary articles to commence housekeeping pending the arrival of funds. He had a consignment of gig-springs for sale, which he thought were worth £30. The second is dated August 5, 1841, asking for acceptance of a prómissory note for £30; to this is appended a copy of the secretary's reply of the same date:

> The Governor directs me to inform you that he cannot have anything to do with money transactions of the kind to which your letter alludes he has therefore directed me to return your note to you, and to enclose you a check for £25, requesting you to repay this sum when it may be convenient for you to do so.

—an exceedingly kind and sympathetic answer.

15. Mathew Moorhouse (about 1813-1876).—Mathew Moorhouse's name was registered on February 4, 1845, M.R.C.S. (England, 1836). Mathew Moorhouse was in practice at Hanley, Staffordshire, where he became acquainted with the Reverend William Ridgway Newland, whom he accompanied to South Australia in the *Sir Charles Forbes*, which arrived at Holdfast Bay in July, 1839. He was then twenty-six years old and had been appointed by the Imperial Government as the first protector of aborigines for South Australia. He had his office on North Terrace, near the present Public Library building. So as to be close to the Newland family, he took up a section at Encounter Bay and built a house and set out a garden on a knoll not far from the Bluff. He had the interests of the natives very much at heart, and in his official capacity on their behalf accompanied Major O'Halloran on his expedition to inquire into the outrages connected with the wreck of the *Maria* on the Coorong, and on the later ones, also under Major O'Halloran, against the Rufus blacks on the Murray. On another occasion he visited Yorke Peninsula with the police to apprehend natives who were afterwards executed for murder. In 1841 he was appointed a justice of the peace, and also became a member of the Statistical Society, to obtain data on the financial condition, resources and prospects of the province. He became a member of the Destitute Board in 1851. Towards the end of 1856 he left for England, where he was engaged for two years in lecturing on emigration to South Australia. Soon after his return he retired from the public service and took up pastoral pursuits, being interested, with others, in properties at Riverton and Saddleworth and near the Hummocks. He managed the latter till 1870, when he sold out his interests and settled at Bartagunya, near Melrose, where he remained till his death, at the age of sixty-three, on October 2, 1862. He was one of the members for the city of Adelaide in the second Parliament, from 1860 to 1862. A portrait of him is in the institute at Melrose, and he was buried in the cemetery near that town. A biographical account, with portrait, appears in "Pastoral Pioneers of South Australia", Volume I, page 88.

16. Robert Montgomery (about 1806-1866).—The name of Robert Montgomery was registered on February 4, 1845, M.R.C.S. (Edinburgh, 1829). Montgomery was killed almost instantaneously by being thrown out of his buggy at Glenelg on March 1, 1866, at the age of fifty-six years. He was buried at Happy Valley Cemetery, "which forms part of the section where he so long resided", the service being conducted by the Reverend C. Manthorpe. (*The South Australian Register*, March 3, 1866, and March 5, 1866, page 2.) The site of this cemetery is now occupied by the Happy Valley Reservoir, the interments therein having been transferred to an adjacent site.

17. Edward Davy (1806-1885).—Edward Davy's name was registered on March 4, 1845, M.R.C.S. (England, 1828), L.A.C. (London, 1827). He was the son of Thomas Davy, a surgeon practising at Ottery St. Mary, Devonshire, and was born on June 11, 1806. Having been deceived into buying a chemist's business in The Strand, London, under the idea that he was purchasing a medical practice, he traded as an operative chemist, dealing mostly in scientific apparatus of his own invention. He also patented "Davy's Diamond Cement" for mending china and glass. In 1836 he published "An Experimental Guide to Chemistry". He also commenced experiments in electric telegraphy, which entitle him to be regarded as one of its pioneers; he invented the "relay". In 1836 he published also "Outline of a New Plan of Telegraphic Communication". In May of that year he unsuccessfully opposed the grant of a patent to Cooke and Wheatstone. In 1838 he patented his "Electro-Chemical Recording Telegraph", and seemed on the way to fame. However, he chose to go to Australia as medical superintendent of an emigrant ship, sailing on April 15, 1839. During his absence his schemes collapsed, and his invention was bought by the Electric Telegraph Company. In Australia Davy started farming and journalism, contributing to *The Argus*, Melbourne, from 1843 to 1845, and then became editor of *The Adelaide Examiner*. From 1848 to 1851 he managed copper smelting works at Yatala. He took charge of the Government Assay Office, Adelaide, in 1852, which he left for a similar appointment at Melbourne at £1,500 a year. This appointment lasted only from July, 1853, to December, 1854, when the post was abolished. He later practised at Malmesbury, in Victoria, where he died on January 27, 1885. In 1884 he was made an honorary member of the Society of Telegraph Engineers and Electricians. His son, H. Davy, M.D., published a memoir of him. (Phillip Mennell: "The Dictionary of Australasian Biography", 1892, page 122. Loyau: "Notable South Australians", 1885, page 113.)

18. George Bull.—The name of George Bull was registered on July 1, 1845, B.M. (Cambridge, no date). There is no record in the State Archives, Adelaide.

19. Thomas Young Cotter (1805-1882).—Thomas Young Cotter's name was registered on January 14, 1846, L. of S.Apoth. (London, 1833). He was born at Bantry, near Cork, and arrived by the *Coromandel* in August, 1836, to take up his position as colonial surgeon. After resigning this position he practised at Adelaide, Mount Barker, Queenstown, Robe and Angaston, and in 1864 he settled in the north. In 1870 he established himself at Port Augusta, where he died on January 9, 1882. There is a biography by the late Dr. A. A. Lendon in the State Archives, Adelaide, and an account of his career appears in Loyau's "The Representative Men of South Australia", 1883, page 82.

20. George Francis Moreton (about 1819-1861).—George Francis Morton's name was registered on April 7, 1846, M.R.C.S. (England, 1844). In 1924 Miss Annette Moreton, of Adelaide, gave to Sir Joseph Verco a number of printed and written documents, twenty-three in number, that had belonged to her father. These Sir Joseph transcribed and annotated (*vide* State Archives, Number 1,009), giving in this way a very good picture of medical education between 1833 and 1844. The series commences with George Morton's indenture (a filled-in, printed form, apparently of general application to any kind of apprentices) to "Joseph D. White, M.D., Surgeon and Apothecary", a very interesting

document, especially in the light of Sir Joseph's commentary. The last document was his registration in South Australia, which contains the first five names on the register, the names of the men constituting the board of that day. Sir Joseph has given a short account of each of these five medical men. A biography by the late Dr. A. A. London, based on Sir Joseph Verco's compilation, is in the State Archives, Adelaide. Moreton practised at Gawler and Watervale, was the first surgeon to the Stockade, and died on November 20, 1861, at Nailsworth.

21. **Henry Chambers.**—Henry Chambers's name was registered on April 7, 1846, L. of C.Apoth. (London, 1843). No record appears in the State Archives, Adelaide.

22. **Ferdinand von Sommer.**—The name of Ferdinand von Sommer was registered on July 7, 1846, M.D. ("Gena" = Jena, no date). No record appears in the State Archives, Adelaide.

23. **Alan M. Thomson.**—The name of Alan M. Thomson was registered on July 7, 1846, M.D. (Glasgow, 1826). No record appears in the State Archives, Adelaide.

24. **Angus James Campbell.**—The name of Angus James Campbell was registered on October 6, 1846, M.R.C.S. (Edinburgh, 1826). No record appears in the State Archives, Adelaide.

25. **Thomas Bayes.**—Thomas Bayes's name was registered on January 5, 1847, L.W.C.A. (London, 1830). There is no record in the State Archives, Adelaide.

26. **Hugh Smith.**—The name of Hugh Smith was registered on January 5, 1847, M.D., M.R.C.S. (Edinburgh, 1842). No record appears in the State Archives, Adelaide.

27. **Lawrence Healey.**—The name of Lawrence Healey was registered on January 5, 1847, M.R.C.S. (London, 1842), L.W.C.A. (London, 1821). No record appears in the State Archives, Adelaide.

28. **John Gemmell.**—The name of John Gemmell was registered on April 6, 1847, M.R.C.S. (Edinburgh, 1837). No record appears in the State Archives, Adelaide.

29. **Robert Waters Moore (1819-1884).**—The name of Robert Waters Moore was registered on July 13, 1847, M.R.C.S. (London, 1842). He was colonial surgeon from 1858 to 1869. He died in 1884, when he was in his sixty-fifth year. His biography has been written by the late Dr. A. A. London under "The Early Protectors of the Aborigines", which is in the State Archives, Adelaide.

30. **Charles John Frederick Bayer.**—The name of Charles John Frederick Bayer was registered on October 5, 1847, M.D. (Bavaria, 1842). Dr. Bayer was born at Munich, in Bavaria, and studied at the University of Erlangen and afterwards at other German universities. He was banished from Bavaria for refusing to disclose information concerning a duel which had come to his knowledge professionally. He arrived in South Australia in 1847 as surgeon in the *Heloise*. He was a great leader of his fellow Germans in South Australia. He married a daughter of Dr. Kent and had a large family, eight surviving him, one son being then in England, intended for the medical profession. He died from apoplexy at North Terrace on August 15, 1867, aged fifty-two years. The Reverend James Jefferis preached a special sermon on his death, in the North Adelaide Congregational Church, and the Press of the day referred to him as a man of large heart and wide beneficence, and wrote a high eulogy of him. At the Exchange on August 21, following his death, a meeting, with Sir J. H. Fisher in the chair, was held to raise a fund, to be called the "Bayer Memorial Fund", for a statue or bust of him, and for the endowment of some benevolent medical institution, or other similar means of perpetuating his memory. (*South Australian Register*, August, 1867, page 2, and August 28, 1867, page 2. Loyau: "Notable South Australians", page 228.) At the inquest on Dr. John Knott (31), he gave a good account of the clinical symptoms after the accident. The bust that was prepared is now in the Art Gallery, Adelaide.

31. **John Knott.**—John Knott, whose name was registered on October 5, 1847, held the qualifications of M.R.C.S. (London, 1829) and L.W.C.A. (London, 1828). He was ship's surgeon in the *Pestonjee Bomanjee*, arriving in October, 1838. On the voyage out, Dr. Knott, the captain and J. C. Hawker rode together on horseback in Tenerife from Santa Cruz to Laguna (J. C. Hawker, "Early Experiences in South Australia", 1899, page 3). In "Papers Relative to South Australia", 1843, page 80, giving statistical returns for the end of the year 1840, John Knott is shown as the owner of Section 308, of 134 acres, bounded on one side by the River Torrens, with one large dwelling house nearly built, one cottage, stable and stockyard. He lived in Hindley Street West. Dr. Knott fell from his horse at Thebarton on Sunday evening, October 20, 1850, and died early next morning from a fracture of the base of the skull, having had a slow pulse and extradural clot. He was attended by Dr. Frederick Charles Bayer, who gave a good clinical account of his symptoms, and by Dr. Kent. Dr. William Wyatt held an inquest on October 21. A verdict of accidental death was recorded. The coroner, in addressing the jury, took the opportunity of referring to and rebutting Press comments on a previous case in which he had not held an inquest, the death being apparently that of Mrs. Duncan, wife of a medical man (3), who had also died as a result of a riding accident. Dr. Knott was forty-four years of age at the time of his death. (*The South Australian Register*, October 22, 1850, page 2.)

32. **William Parr Hill.**—The name of William Parr Hill was registered on October 5, 1847, M.R.C.S. (London, 1846). From December, 1860, till the end of 1865, Dr. Hill was medical officer at Encounter Bay. (C. R. Hodge, "Encounter Bay", undated, page 51.)

33. **George Lawson (1811-1846).**—The name of George Lawson was entered in the register on October 5, 1847, F.M. *et* Chir. (Glasgow, 1835). George Lawson, the fifth son of Stephen Lawson, merchant and banker, was born at Edinburgh on December 11, 1811. He came to South Australia as surgeon to the ship *Siam*. He was appointed, on September 13, 1844, to succeed Richard Penney at Port Lincoln, at a salary of £40 a year. Here he served the Government for twenty-two years, dying on February 10, 1867, at Ravelstone, Port Lincoln, from the effects of a fall from his horse. He left a wife and six children. A bold and fearless rider, he went long journeys to visit the sick. Generous to a fault, he visited the shepherd's hut as freely as the homes of the well-to-do. (*The South Australian Register*, March 28, 1867, page 7.)

34. **George Bryden.**—The name of George Bryden was registered on January 4, 1848, M.R.C.S., L.W.C.A. (London, no dates). No record appears in the State Archives, Adelaide.

35. **Thomas Taylor.**—The name of Thomas Taylor was registered on April 18, 1848, M.R.C.S. (London, 1841). Dr. Thomas Taylor was elected Mayor of Kensington and Norwood in 1861 and again in 1863. He lived in High Street, Kensington, in the residence afterwards occupied successively by Dr. Benson, Dr. Sprod and Dr. Borthwick. He was living in England "at a very advanced age" in 1903. (Gordon and Morris: "Fifty Years' History of the Town of Kensington and Norwood", Adelaide, 1903; portrait on page 40, text on page 45.)

36. **Frederick Emil Renner.**—Frederick Emil Renner, M.D. ("Gena" = Jena, 1847), whose name was registered on July 5, 1848, was in Adelaide in the early fifties. Later he was the first practising physician in the Wentworth district (1857-1870). He returned to Adelaide in 1870 and was government medical officer to the overland telegraph parties (1871-1872). A photograph of him appears in "Exploring in the Seventies", by Alfred Giles. Renner Springs, between Tennant Creek and Powell Creek, was named after him.

ILLUSTRATIONS TO THE ARTICLE BY DR. RUPERT A. WILLIS.

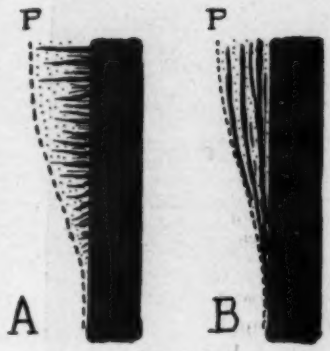

FIGURE I.

Diagram illustrating the two types of new bone formation by expanded periosteum. *A* = radial spines, *B* = "onion-skin" laminæ, *P* = periosteum. Tumour shown stippled.

FIGURE II.

Skiagram (positive print) of the shaft of the femur, showing "onion-skin" laminæ in metastatic neuroblastoma. *P* = the most recently formed periosteal bone.

FIGURE III.

Skiagram of a piece of skull, showing bony spines projecting in metastatic neuroblastoma on both inner and outer surfaces of the bone.

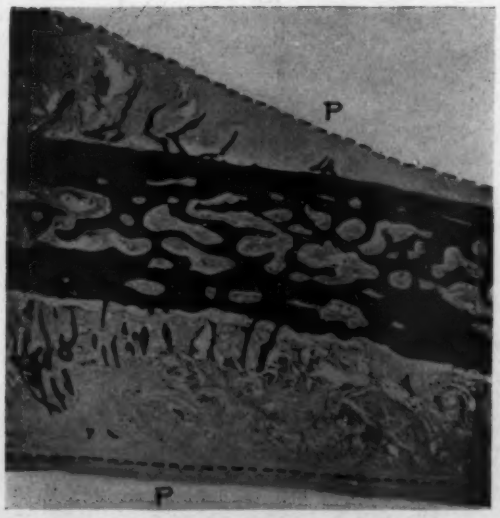

FIGURE IV.

Photograph of a longitudinal section of part of a rib affected by metastatic neuroblastoma. The bony tissue is in black, the radial bony spurs projecting from the rib being clearly shown. *PP* = the periosteum expanded by the growth. (×7.)

FIGURE V.

Skiagram of osteogenic sarcoma of femur, showing both laminæ (*L*) and coarse radial spurs (*S*) of new bone formed by expanded periosteum.

ILLUSTRATIONS TO THE ARTICLE BY DR. RUPERT A. WILLIS.

FIGURE VI.

Photograph of microscopic section of the tumour of Figure V, showing one of the bony spurs projecting inwards from the expanded periosteum (*PP*). *T* = tumour tissue. Note that the bony spur in this case has its base at the periosteum and its apex pointing inwards towards the bone. (×10.)

FIGURE VII.

Ossifying pulmonary metastases from osteogenic sarcoma.

FIGURE VIII.

Osteogenic sarcoma of scapula supervening on Paget's disease. Notice the greatly thickened clavicle.

FIGURE IX.

Central fibrosarcoma of the tibia.

ILLUSTRATIONS TO THE ARTICLE BY DR. RUPERT A. WILLIS.

FIGURE X.

Recurrent fibrosarcoma in the amputation stump.

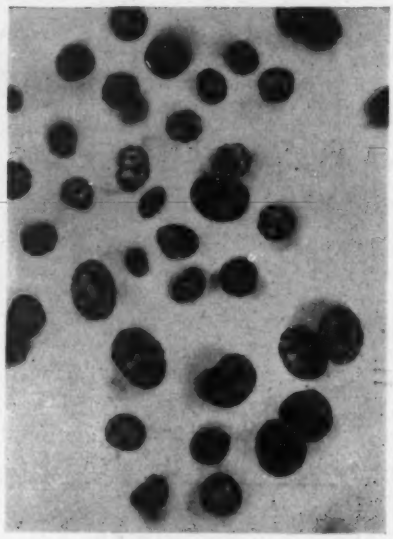

FIGURE XII.

Photomicrograph of smear of myelomatous tissue, showing typical plasma cells. (x 1,000.)

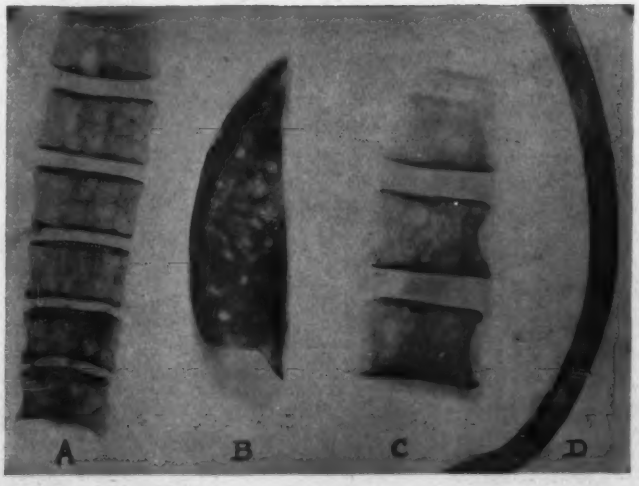

FIGURE XI.

Lesions of multiple myelomatosis seen in skiagrams of bones obtained *post mortem*. *A* = thoracic vertebræ, *B* = ilium, *C* = lumbar vertebræ, *D* = rib.

ILLUSTRATIONS TO THE ARTICLE BY DR. EDWARD FORD.

FIGURE II.
Artificially deformed skull from the Arawe district, New Britain.

FIGURE III.
Infant from a village near Gasmata, New Britain, showing method of head binding to produce cranial distortion.

FIGURE IV.
Mother and infant, New Britain, showing characteristic conical cranial deformation.

FIGURE V.
Mother and infant, New Britain, showing cranial deformation. The outline of the woman's head is obscured by the method of hairdressing.

37. John Travis Leake.—The name of John Travis Leake was registered on July 5, 1848, M.D. (Kiel, 1841). No record appears in the State Archives, Adelaide.

38. Matthew H. S. Blood.—There is no record in the State Archives, Adelaide, concerning Matthew H. S. Blood, whose name was registered on July 5, 1848. He held the M.R.C.S. (England, 1831).

39. Charles Houlton Webb.—The name of Charles Houlton Webb was registered on January 2, 1849, L.W.C.A. (London, 1845). No record appears in the State Archives, Adelaide.

40. Richard Eades.—The name of Richard Eades was registered on January 2, 1849, M.B. (Dublin, 1836), F.R.C.S. (Ireland, 1844), M.R.C.S. (London, 1834). In Dr. A. A. London's notes it is stated that he reached Adelaide in 1849, and Melbourne in 1851.

41. William Hillebrand.—The name of William Hillebrand was registered on April 3, 1849, M.D. (Berlin, 1844). No record appears in the State Archives, Adelaide.

42. James Phillips.—The name of James Phillips was registered on April 3, 1849, M.R.C.S., L.W.C.A. (London, 1843). James Pryce Phillips left Saint Bartholomew's Hospital to come to South Australia in 1843. At Saint Bartholomew's he got to know intimately Dr. Wylde, who had entered that hospital in 1840 and who came out to Australia in 1850 and practised at The Semaphore. Phillips was a house surgeon at the Adelaide Hospital "as far back as 1868" (*sic*). He then engaged in private practice, which became extensive. He is described as being of a quiet and retiring disposition. He and his wife lived in an old-fashioned thatched cottage on North Terrace. He retired from professional work during the palmy days of Broken Hill mining, about fifteen years before his death at North Terrace on April 21, 1903. (His age is not given in the death notice.) He was then the oldest medical practitioner in Adelaide, being succeeded in this by the late Dr. T. W. Corbin. For a number of years he was an honorary surgeon at the Adelaide Hospital and was also a member of the board of management of the hospital. (*The Adelaide Observer*, April 25, 1903, page 21.)

43. John Forster.—The name of John Forster was registered on April 3, 1849, M.R.C.S., L.W.C.A. (London, 1830). Dr. Forster came to South Australia from Yorkshire in 1849. After practising his profession for some time he took a large consignment of horses to India for army purposes. Soon after his return, he went to New Zealand for two years. Coming back to South Australia, he engaged in pastoral pursuits, but meeting with reverses, returned to professional work, which he continued till three years before his death, at Hackney, in November, 1891, in his eighty-fifth year. He lived for a great part of his time in the colony in the Mount Barker district, and for a while at Hindmarsh. He had no family. (*The Adelaide Observer*, November 21, 1891, page 29.)

44. Daniel Baruh.—The name of Daniel Baruh was registered on July 11, 1849, M.D. (Aberdeen, 1816). The *Morning Chronicle* of January 31, 1846, is quoted as follows:

> In 1831, Dr. Baruh, who holds his diploma from Aberdeen, was authorized by Royal Ordinance to practise in France. Last year he was sentenced to six months' imprisonment, 500 fr. fine, and 1000 fr. for damages, for having administered an arsenical preparation which caused his patient's death. The "Moniteur" now publishes a report from the Minister of Instruction to the King—"Previously to the affair in question, serious complaints had been made of this person and they were evidently well founded. I have therefore the honour to submit to Your Majesty the Draft of an Ordinance revoking the authorization granted to Mr. Baruh. Inasmuch as France is liberal in the reception of learned foreigners worthy of her hospitality, she is bound to be severe towards those who do not merit to be adopted by her—Galignani."

Dr. Baruh died suddenly at Mount Pleasant on June 6, 1875, aged eighty-five years. During his residence in the eastern and north-eastern districts of the province he held various positions in connexion with friendly societies and the Government.

45. Henry Scott.—Henry Scott, whose name was registered on July 11, 1849, held the M.R.C.S. (London, 1832), and later obtained the F.R.C.S. (England, 1854). No record appears in the State Archives, Adelaide.

46. Robert Philips.—The name of Robert Philips was registered on July 11, 1849, M.R.C.S. (London, 1830). No record appears in the State Archives, Adelaide.

47. Charles Lloyd.—Charles Lloyd's name was registered on October 2, 1849, M.R.C.S., L.W.C.A. (London, no dates). There is no record in the State Archives, Adelaide.

48. Richard William Schmidt.—The name of Richard William Schmidt was registered on October 2, 1849, M.D. (Berlin, 1847). No record appears in the State Archives, Adelaide.

49. John Smith.—John Smith, whose name was registered on October 2, 1849, held the M.R.C.S. (Edinburgh, 1835). No record appears in the State Archives, Adelaide.

50. Richard Gardener Jay.—The name of Richard Gardener Jay was registered on October 2, 1849, L.W.C.A. (London, 1837). Jay was born at Ipswich, Suffolk, in which county his ancestors had lived for centuries. After being apprenticed, he entered Guy's Hospital and qualified later as M.R.C.S. Though possessed of considerable independent means, he emigrated to South Australia in the late forties ("early forties" appears erroneously in "Adelaide and Vicinity", 1901). On landing in Adelaide, he remained for some time in the city and then settled at Willunga, where he practised for over thirty years, acquiring an extensive practice. He became a wealthy man, but in later years lost considerably in mining speculations. He was early made a justice of the peace. His son, William Eugene Jay (registered number 195), who was in medical practice at Wallaroo, died suddenly and his father took up the practice and remained at Wallaroo until he became ill in 1878, when he was removed to Adelaide, where he died. ("Adelaide and Vicinity", 1901, page 463, where the second Christian name is spelled "Gardiner".)

51. Edward Wehl (about 1823-1876).—The name of Edward Wehl was registered on October 2, 1849, M.D. (Hanover, 1848). Dr. Wehl was a German by birth and came to Mount Gambier in 1849 as a young man of about twenty-five years, before the town itself actually existed, the country being in the hands of squatters only. With the exception of the time when he and the rest of the population went to the Victorian diggings in the fifties, he resided for the rest of his life at Mount Gambier. He married a sister of Baron von Mueller, and she, with twelve children out of fifteen, survived him. He died at the age of fifty-three, on February 11, 1876, from hydatid disease of the lungs and abdominal cavity; this was at first mistaken for dropsy and was recognized only after tapping. (*The South Australian Register*, February 2, 1876, page 14.)

52. William Horatio Scholl (about 1809-1876).—The name of William Horatio Scholl was registered on October 2, 1849, no qualifications being entered. In Johns's "Notable Australians" he is said to have had the M.R.C.S. Dr. Scholl in his earlier years was a surgeon in the Spanish army. He arrived in South Australia in September, 1849, from Western Australia, where he had been living for several years and had been proprietor of a newspaper. He began practice in North Adelaide, but shortly afterwards moved to South Adelaide and remained there till 1860. He then went to Wallaroo, where he remained till his death from Bright's disease, after an illness of three months, in his sixty-eighth year, on November 29, 1876, leaving a widow, three sons and two

daughters. The late Mr. L. H. Scholl, Under-Secretary and Government Statist of South Australia, was a son. During his last three years at Wallaroo he was resident surgeon at the Wallaroo Hospital. *The Wallaroo Times* of December 2, 1876, published an appreciation of his services and kindness to the community. (*The South Australian Register*, December 2, 1876, page 14, and December 30, page 16.)

Names Absent from the Register.

The names of the following men do not appear in the medical register, either because they had died or left the province before registration was required, that is, before the end of 1844, or because they did not practise their profession, at least not sufficiently to warrant registration.

Edward Wright, M.D.—Dr. Edward Wright was medical officer to the Survey Department. He arrived at Kangaroo Island, as surgeon to the *Cygnet* on September 11, 1836. His story is told by "Austin Harding" (the late Dr. A. A. Lendon) in *The Adelaide Medical Students' Society Review*, Volume XIX, Number 31, June, 1927, page 44.

W. H. Leigh.—Dr. W. H. Leigh, "late surgeon of the Australian Company's ship *South Australian*", wrote "Reconnoitering Voyages and Travels with Adventures in the New Colonies of South Australia, a Particular Description of the Town of Adelaide and Kangaroo Island; . . . During the Years 1836, 1837 and 1838" (1839). He arrived at Kangaroo Island in April, 1837, and spent apparently four or five months in South Australia.

Charles George Everard (1794-1876).—Dr. Charles George Everard arrived by the *Africaine* in November, 1837. Under the title "Dr. Charles George Everard, Physician and Farmer", Dr. A. A. Lendon gives a full account of him in *The Royal Geographical Society of Australasia, South Australian Branch, Proceedings*, Volume XXXII, 1930-1931, page 28.

John Rankine.—Dr. John Rankine arrived in 1839, settled at Strathalbyn, and was a pioneer of Hindmarsh Island. (C. R. Hodge: "Encounter Bay", undated, published 1933, page 69.)

Richard Penney.—Dr. Richard Penney came out as surgeon on the *Branken Moor*, and in July, 1840, was attached as surgeon to the United Fishing Company at Encounter Bay. On April 13, 1843, he went to Port Lincoln to succeed Dr. Harvey, who had died. Dr. Penney died on January 15, 1844, and was succeeded at Port Lincoln by Dr. George Lawson (33). The pathetic story of the troubles of Dr. Richard Penney is told by the late Dr. A. A. Lendon in *The Royal Geographical Society of Australasia, South Australian Branch, Proceedings*, Volume XXXI, 1929-1930, page 20. See also C. R. Hodge, "Encounter Bay", page 49.

R. G. Jameson.—Dr. R. G. Jameson described himself as "Late Surgeon Superintendent of Emigrants to South Australia" in a book he wrote, entitled "New Zealand, South Australia and New South Wales: A Record of Recent Travels in these Colonies", London, Smith, Elder and Company, undated (about 1842). He reached Adelaide in the *Surrey* in October, 1838. His book contains an account of Adelaide.

Lovell Byass.—Dr. Lovell Byass was surgeon to the settlement at Kingscote, Kangaroo Island, up to the end of 1838, when he was succeeded by Dr. J. B. Harvey.

James Benjamin Harvey.—Dr. James Benjamin Harvey, M.R.C.S., was in the employ of the South Australian Company from February, 1839, to October 31, 1842. *The Government Gazette* of June 30, 1839, announced his transfer to Port Lincoln as collector of customs and also postmaster and port officer. Dr. Harvey was a corresponding member of the Zoological Society of London. In *The South Australian Magazine* for December, 1841, is an article by him on the natural history of the Port Lincoln district. The statement that he died in agonising torture from a surfeit of water-melons probably really means from intestinal obstruction. This was in 1843. He was buried on Boston Island, where his wife also was buried; but there is no tombstone or enclosure to mark the site. (Mrs. Watts: "Family Life in South Australia", page 56. *The Adelaide Examiner*, March 15, 1843, page 2. A. A. Lendon: "A Link between Kangaroo Island and Port Lincoln, J. B. Harvey, M.R.C.S.", *The Port Lincoln Times*, March 9, 1928.)

M. McShane.—Dr. M. McShane was surgeon in the *Katherine Stuart Forbes*, which left Holdfast Bay for England on April 11, 1840. (J. C. Hawker: "Early Experiences in Australia", 1899, page 69.)

M. H. Garde.—Dr. M. H. Garde was surgeon in the *Siam*, which left Portsmouth on December 12, 1840, and reached Holdfast Bay on April 25, 1841. (J. C. Hawker: *loco citato*, page 73.)

McDougall.—Dr. McDougall, manager of Malcolm's station at Poltalloch, some time after 1841, is mentioned by Inspector Alexander Tolmer in "Reminiscences of an Adventurous and Chequered Career", Volume I, 1882, page 240. He married Tolmer's sister-in-law, Catherine Carter, later left Australia, and was in a shipwreck at Honolulu.

Marslin.—Dr. Marslin, of Murray Bridge, who was in South Australia apparently some time after 1841, is also mentioned by Inspector Tolmer (*loco citato*, Volume I, page 241).

De Lisle.—Inspector Tolmer (*loco citato*, Volume II, page 51) mentions meeting at Hobart in December, 1846, Dr. De Lisle and Lieutenant McGill, officers of the regiment stationed at Hobart, whom he had known intimately when the detachment had been quartered at Adelaide.

Addison.—Dr. Addison is stated by Inspector Tolmer (*loco citato*, Volume II, page 89) to have been living at the Reedbeds, near Adelaide, in 1848.

David Mahony.—Dr. David Mahony, of County Kerry, Ireland, settled at Gawler at the end of the forties and married Elizabeth Sarah Reid. The name was also spelt "Mahoney". Nearly the whole of Gawler East was known as Mahony's Paddock. He called the house he built "Yenda". He was the first worshipful master of the Lodge of Fidelity. ("The First Settlers at Gawler: Reminiscences of Mrs. Mahony", edited by the late Dr. A. A. Lendon, *The Royal Geographical Society of Australasia, South Australian Branch, Proceedings*, Volume XXVIII, reprint with additions, page 29.)

Reports of Cases.

ACUTE IODIDE ERUPTION FOLLOWING THE INGESTION OF A SMALL AMOUNT OF POTASSIUM IODIDE.

By Ivan M. Wartski, M.B., B.S.,
Clinical Assistant to the Dermatological Department, Royal Melbourne Hospital, Melbourne.

Clinical History.

P.R., a male, aged sixty-six years, an old-age pensioner, had a suprapubic cystostomy performed two and a half years ago, on account of retention of urine and dilatation of the bladder, the result of urethral obstruction by an enlarged prostate gland. He thereafter had a catheter permanently fixed into the bladder suprapubically. He had also been treated for varicose veins, ulcers and eczema of

the legs. He had complained of some dyspnœa on exertion four years prior to his operation. Renal function tests were carried out soon after the operation. The results were as shown in Table I.

TABLE I.
Urea Concentration Test.

Time of Estimation.	Urea Percentage.
Before meal	1·05
First hour	1·25
Second hour	1·80
Third hour	1·00

The urea content of the blood was estimated at 40 milligrammes per 100 cubic centimetres.

On May 30, 1938, the patient was referred to the dermatological department, complaining of an eruption on the face, neck and hands, which had been present for two days. It had occurred within twenty-four hours of his having taken three doses only of a mixture containing potassium iodide and stramonium (Royal Melbourne Hospital Pharmacopœia). This contains only 0·3 gramme (five grains) of potassium iodide to each dose. It had been ordered for a winter cough. On the first appearance of the eruption the patient ceased taking the mixture.

On examination he presented a remarkable appearance (see Figure I). His features were quite leonine on account

FIGURE I.
P.R., aged sixty-six years, showing acute iodide eruption.

of tumefaction, and his whole face was closely covered with discrete vesico-pustules, varying in size from that of a millet seed to the size of a threepenny piece. Both lips, especially the lower, were greatly swollen. His conjunctivæ were congested, and on the nape of the neck were similar vesico-pustules, even more numerous than on his face. Some had become confluent to produce lakes of turbid fluid. On the dorsum of his right hand was a hæmorrhagic bulla about three centimetres by two centimetres in area. The sweat furrow of his back and his shoulders bore discrete follicular pustules.

On June 2, 1938, he was admitted to hospital and was examined. His temperature was 36·2° C. (97° F.), his pulse rate was 78 beats to the minute, and his respirations numbered 22 to the minute. His systolic blood pressure was 140 and his diastolic pressure 90 millimetres of mercury. His tongue was furred. He had a complete set of artificial dentures and his gums were healthy. His throat was also healthy. Examination of the heart revealed that the apex beat was in the fifth intercostal space, 9·4 centimetres (three and three-quarter inches) from the middle line. No right-sided cardiac dulness was present. The heart sounds were regular but faint. The lungs were normal. The abdomen was protuberant, but there was no enlargement of the liver or spleen. The suprapubic catheter was *in situ*. The deep reflexes were present.

No organisms were grown on attempted culture from the blood. The specific gravity of the urine was 1,014; it was alkaline, and contained a large quantity of albumin, but no sugar, acetone or diacetic acid.

Renal function tests were carried out. The results were as shown in Table II.

TABLE II.
Urea Concentration Test.

Time of Estimation.	Volume of Urine in Cubic Centimetres.	Urea Percentage.
Before meal	153	0·60
First hour	90	0·70
Second hour	130	0·50
Third hour	78	0·60

The amount of urea excreted in three hours was 1·75 grammes. The urea content of the blood was estimated at 35 milligrammes per 100 cubic centimetres.

After the patient's admission to hospital further hæmorrhagic bullæ developed on the dorsum of both hands, and the facial lesions became crusted before they finally retrogressed. Treatment consisted mainly of the administration of sodium chloride in capsules three times a day to hasten elimination. The dose used was 1·3 grammes (20 grains). Fluids were also given freely. Calamine lotion, to each ounce of which 0·03 gramme (half a grain) of perchloride of mercury had been added, was applied locally. Sodium thiosulphate was given intravenously and by mouth. The general condition of the patient gradually became worse, and on July 9, 1938, he died. *Post mortem* examination revealed pyonephrosis and bronchopneumonia. The eruption had not entirely disappeared at the time of his death.

Comment.

Adamson,[1] in *The British Journal of Dermatology and Syphilis*, quoting Morrow, makes the following statement:

> In certain drug eruptions and particularly the bullous iodide eruptions there was a proneness for these eruptions to develop especially in connection with cardiac and renal disorders.

In the same article he publishes notes of a patient with malignant endocarditis and renal disease; a single dose of 0·03 gramme (five grains) of iodide evoked a severe iodide eruption in this case. He points out that this would seem to exclude defective elimination as a sufficient explanation; and further, that the commonly fatal issue in these cases may be due, at any rate in part, to the constitutional disturbances accompanying the eruption, as evidenced by the occurrence of fatal iodide eruptions even in the absence of cardiac and renal disease.

Sequeira[2] makes the following statement:

> Eruptions caused by iodides are most commonly seen in patients suffering from grave renal and cardiac disease, but occasionally there is a remarkable idiosyncrasy apart from conditions of defective elimination.

Roxburgh,[3] writing of drug eruptions and referring to iodide, remarks as follows:

> Vesicular and bullous (iodide) eruptions may also occur, especially in patients with renal disease or infective endocarditis.

Summary.

1. A case of vesico-pustular and bullous iodide eruption is described.

2. It occurred as a result of the ingestion of a very small amount of potassium iodide, three doses of 0·3 gramme (five grains) each.

3. Attention is drawn to the association of acute iodide eruption with grave cardiac and (as in this case) renal diseases.

4. These subjects apparently have a peculiar idiosyncrasy to iodides, which is not explained by defective elimination.

5. A fatal termination is unusual in these cases. It is probably contributed to by constitutional disturbances accompanying the eruption.

Conclusions.

It would seem that iodides should be prescribed with extreme caution in the presence of gross cardiac and renal disease; and that blood mixtures and tonics advertised in the lay Press, the majority of which contain iodides, may be the cause of serious eruptions in, and should be eschewed by, sufferers from cardiac and renal diseases.

Acknowledgement.

I wish to thank Dr. R. R. Wettenhall, senior dermatologist to the Royal Melbourne Hospital, for permission to investigate this patient and publish these notes.

References.

(1) H. G. Adamson: "Bullous Iodide Eruption in Association with Endocarditis and Nephritis", *The British Journal of Dermatology and Syphilis*, April, 1938, page 167.
(2) J. H. Sequeira: "Diseases of the Skin", Fourth Edition, 1927, page 336.
(3) A. C. Roxburgh: "Common Skin Diseases", Second Edition, 1934, page 93.

Reviews.

ANÆSTHESIA FOR NURSES.

Much valuable information, set out in a very concise way, is contained in J. K. Watson's short book, "Anæsthesia and Analgesia for Nurses and Midwives".[1] On the whole the ground is covered thoroughly, but unfortunately the author departs from his originally stated intention of providing an elementary survey and proceeds to a discussion of technical detail which is neither of primary interest nor of great help to his readers. The result is that much controversial matter is included, tending to obscure essential points, and there are many omissions which otherwise might have been avoided. There is also apparent throughout the volume a conservative bias which, while generally admirable, has led to the entire absence of any reference to valuable improvements like the Magill endotracheal tube and the carbon-dioxide absorption technique, while anachronisms like the "A.C.E." mixture and the use of "colonic ether" by the vapour method are described with evident approval.

The subject matter is set out in ten chapters, which cover consecutively general principles, inhalational agents (four chapters), spinal, intravenous, rectal and local methods, and finally normal labour. A good examination of the legal responsibilities of those concerned in the induction of anæsthesia is included, and repeated emphasis is laid on the necessity for preserving at all times a clear airway. Particular attention is also directed to the respiratory depression which excessive premedication may produce. The necessity for extreme care in the assembling of apparatus is well stressed; but the Junker's apparatus, illustrated on page 43, certainly seems to be wrongly connected. Statements to which exception may be taken are that anæsthetic vapours irritate the muscles of the upper respiratory passages, so producing spasm; that a lack of oxygen in the tissues constitutes anoxæmia; that transnasal tracheal intubation and pharyngeal packing are not desirable procedures; that induction with cyclopropane proceeds much on the same lines as with nitrous oxide (when it is the reverse); and so on.

The final chapter, on anæsthesia and analgesia in normal labour, is the chief virtue of this book. The various considerations involved are adequately examined and the procedures recommended are well dealt with under the three headings of anæsthesia, analgesia and amnesia. Chloroform receives its usual commendation; "Avertin" in its "obstetric" dosage of 75 milligrammes per kilogram is favourably discussed, and nitrous oxide, with oxygen is described as an ideal method. In connexion with domiciliary work, an enthusiastic and rather optimistic account of nitrous oxide and air analgesia, produced by the Queen Charlotte's gas-air apparatus, is presented, and various models of the machine are described and illustrated. Scopolamine-morphine narcosis is considered in detail, but is not so favourably regarded as the alternative methods.

[1] "Anæsthesia and Analgesia for Nurses and Midwives", by J. K. Watson, M.D.; 1938. Bristol: John Wright and Sons Limited; London: Simpkin Marshall Limited. Crown 8vo, pp. 135, with illustrations. Price: 3s. 6d. net.

THE SEASONAL PERIODICITY OF MALARIA.

Despite the wealth of literature on malaria in all its aspects, several gaps in our knowledge still remain. Clifford A. Gill's monograph "The Seasonal Periodicity of Malaria" has helped to fill at least one hiatus.[1]

Though the author's classification of epidemiological types of the disease into temperate, subtropical, tropical and equatorial zone forms is purely arbitrary, it yet provides a useful basis of study. The well-known periodicity of malaria has for the most part been regarded as a result of the transmission factor—a combination of the anopheline and meteorological influences—but, as shown by Dr. Gill, this hypothesis fails in many instances to explain the facts. Many of the seasonal and epidemic waves are shown to commence before any increase in the number of mosquitoes. The most important determining cause, according to Dr. Gill, of these seasonal waves of increased malarial morbidity and mortality is the relapse factor. This seasonal relapse, for which no cause is as yet known, must be distinguished from the clinical non-seasonal relapse, which may be precipitated by cold, acute illnesses, general anæsthetics *et cetera*, though the precise way in which these cause reactivation is uncertain. The seasonal relapse, and not new infections, initiates the seasonal wave and to a large extent determines its magnitude. As shown by Dr. Gill, spring epidemics are always preceded by an increased autumnal wave, while it is important to remember that, especially in the malaria of temperate zones, there may be a latent period of thirty to forty weeks between the time of infection and the appearance of clinical manifestations. Having arrived at the conclusion that the relapse factor is of primary importance, the author is at a loss to discover the factors concerned in its causation. Hence the reader feels that he has gained a possible answer to one question, only to be faced with another problem, more difficult of solution.

It is unfortunate that the author has been unable to collect sufficient data from the southern hemisphere. We suggest that it might be possible to utilize our resources in northern Australia, Papua and New Guinea, to elucidate further this interesting aspect of malaria.

[1] "The Seasonal Periodicity of Malaria and the Mechanism of the Epidemic Wave", by C. A. Gill, M.R.C.P., M.R.C.S., D.P.H., D.T.M. and H.; 1938. London: J. and A. Churchill Limited. Demy 8vo, pp. 147, with a map and 17 illustrations. Price: 10s. 6d. net.

Notes on Books, Current Journals and New Appliances.

PROGRESS IN ENDOCRINOLOGY.

During the last decade the study of endocrinology has increased with such rapidity that it is difficult for most to keep pace with accumulating facts and changing theories. For this reason a recent issue of *The Prescriber*[2] which has been devoted to reports of recent progress in endocrinology should be welcomed. This special number includes chapters devoted to the thyreoid and parathyreoid glands, the adrenal and pituitary glands, the pancreas, the female reproductive system and the male gonad. It affords an excellent survey of the important developments that have taken place in these fields during the past year.

[2] *The Prescriber*, May, 1938.

The Medical Journal of Australia

SATURDAY, OCTOBER 29, 1938.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

HEALTH WEEK.

"HEALTH and good estate of body are above all gold", says the writer of Ecclesiastes; in New South Wales an attempt is now afoot to remind the public of this fact by the holding of the annual function, Health Week. Few medical men and fewer laymen realize the amount of organization and the expense involved in telling our citizens that preventible disease may be prevented and avoidable disease avoided by the exercise of intelligence and common sense, and that it is still possible to make life a happier business for all of us.

Great praise is due to the New South Wales Department of Health for the work accomplished in this matter. From the office of the Director-General of Public Health have been issued, in round numbers, some twenty booklets and leaflets, which anybody may have for the asking. These carefully compiled and well-written pamphlets deal in plain language with many aspects of preventive medicine and with the recognition of, and the suitable treatment for a wide variety of infectious diseases. Today no intelligent person can plead ignorance of the means employed to combat the spread of scarlet fever, typhoid fever, whooping cough or the common cold. The householder who wishes to circumvent the wily rat or to rid his home of flies, fleas, cockroaches or mosquitoes has nobody but himself to blame if he cannot encompass these tasks; the Board of Health will give him the necessary advice in print, and if he can read he has but to follow the rules.

Specially to be commended to the notice of the public is the small book issued by the National Safety Council of Australia, and bearing the title "Safety First". This is a manual dealing with most of the emergencies and accidents that call for quick treatment in the home, the factory, the street or the playground. Compiled by Mr. A. G. White, this book has already had a circulation of ninety thousand copies. Our best wish for its success is that future issues of this or similar works may be found in all Australian homes.

During Health Week every effort is exerted to arouse the public to a sense of communal responsibility. Many of us think little about the problems of health and disease until we fall sick ourselves; patients and their relatives regard sickness only as a problem for the individual. But in a good democracy we must all subscribe to the gospel that upon a man himself, his surroundings, his knowledge of hygiene and his respect for the rules of sanitation depends, in the long run, the health of all his fellows.

Apart from the neglect of many people to treat their neighbours as they would themselves in matters of public health, this community, in a country filled with all the good things of the earth, is confronted with another great problem. Any housewife knows that the prices charged in Australia for many essential foods are beyond all reason and smack more of uncontrolled piracy than of fair trade. There must be something wrong, for instance, with a system under which a purchaser may buy Victorian cream in Sydney at a rate lower than that asked for the local product. It is common knowledge also that many children of the poorer class do not receive the minimum quantity of milk

necessary to avoid deficiency diseases; their parents simply cannot afford to buy in sufficient amount the most necessary of all foods for the young. Much the same may be said of our supplies of fresh fruits and vegetables; yet at certain seasons one may witness, after a short motor trip from Sydney to the fruit-growing districts, the sad spectacle of heaps of oranges rotting upon the ground. Marketing costs and other charges raise obstacles, so far insuperable, between producer and consumer. In South America thousands of tons of coffee have been burnt as fuel for steamers and locomotives, or thrown into the sea. So strange are the laws of modern economics—if there are any laws—that in most instances the more plentiful a useful commodity is, the harder it is to procure.

Since there is a regrettable tendency in most of us to think more of the individual than the collective good, the Director-General of Public Health in New South Wales has been forced to point out that further powers of compulsion than he at present possesses in matters of public health administration are urgently needed. Dr. Morris says that certain large municipalities enjoying large revenues refuse to employ health inspectors. He pleads for greater cooperation between his department and the local authorities; but that cooperation apparently is not always forthcoming. Apparently, too, through pressure of parliamentary business, it is at present impossible to make vital amendments to the *Public Health Act* in order to bring certain recalcitrant councils to their senses. The Director tells us further that the immunization campaign against diphtheria has been so far an unqualified success, a statement patently true. Thus far, in New South Wales, 150,000 children have been immunized against the disease; "... but if the disease is to be effectively controlled, another 150,000 should be treated and the number yearly should be about 50,000. With from 300,000 to 400,000 immunized, there would be a big drop in the diphtheria figures." But because compulsory immunization would raise an outcry from all the cranks in the land, Dr. Morris thinks it impracticable to enforce it in this or any other British community. With all respect, why so? In many matters the doings of the level-headed and the cranks alike are controlled by statute. No crank, whatever his objections, can avoid the obligation to pay taxes when due, to obey the traffic laws, to find the money for his wireless licence, or to vote at an election. Surely the cranks, if they try to shelve their duty to the community, can be forced by act of parliament to safeguard the lives of their own children and those of their neighbours.

Current Comment.

ORGANIZING RHEUMATIC SERVICES.

An immense literature on the subject of the rheumatic and rheumatoid diseases is accumulating. Extensive investigations of rheumatism are being undertaken all over the world. These exhaustive studies are justified in view of the extreme suffering and crippling caused by these diseases, and the consequent burden thrown upon industry and the vast economic loss entailed. A recent contribution to the subject has been made by H. L. Bacal and R. R. Struthers.[1] They inform us that the rheumatism of childhood has been a serious problem in Montreal for many years. They state that the temperate climate, marked by sudden changes in weather, the social and environmental conditions of the poorer classes, and possibly the type of homes occupied by unfortunate children, have made Montreal one of the largest centres of juvenile rheumatic infection on the continent of America. The authors consider that the crippling effects of rheumatism are comparable with those of such other chronic diseases as syphilis and tuberculosis. They believe that, once it becomes chronic, rheumatism behaves in a similar fashion to tuberculosis and should be treated in the same manner. Attacks of the various manifestations of the rheumatic cycle may recur periodically, and one attack predisposes to another. Their experience is that most bouts occur in the spring, and that females are more prone to the infection than males. They separate rheumatic children into four groups. There is first the "unwell" child, poor in physique and prone to recurrent respiratory infections and to "growing pains". He may manifest no definite evidence of rheumatic infection, but he is a potential candidate for rheumatism, especially when a family history of the disease exists. The second group includes those who suffer from Sydenham's chorea. Next come those affected with acute rheumatic fever, which is not usually characterized by severe arthritis in children, but by evidence of inflammation of the periarticular tissues and muscles. The arthritis is migratory and does not lead to permanent disability or suppuration of the joints. The

[1] *The Canadian Medical Association Journal*, March, 1938.

fourth group contains those afflicted with rheumatic carditis, which is the most common and severe complication of rheumatism and a menace to the future of a rheumatic child. It is held that from 40% to 50% of rheumatic patients eventually suffer from some cardiac lesion.

Bacal and Struthers were able to institute a "rheumatism service" at the Children's Memorial Hospital at Montreal, where patients were observed, if possible, from the onset of the disease, were admitted as in-patients, and, when discharged to their homes, were seen periodically in the outdoor department at the cardiac clinic. Sufferers from acute rheumatism are first admitted to the main hospital and isolated in cubicles until it is certain that no complicating infectious condition exists, whereupon they are transferred to the rheumatism pavilion. The pavilion has a balcony across the front, large enough to permit all the children to be out of doors when the weather allows. It is well lighted and well ventilated. A temperature of 70° to 72° F. is constantly maintained, with a relative humidity of 45% to 55%. There are certain cubicles to which new patients are admitted. Those suffering from active carditis, pronounced chorea or an infection of the upper respiratory passages are often placed there. The ward is bright and attractive. The beds are painted a soft green, while the bedside tables, the chairs and the walls are light cream in colour. On admission each child is weighed, measured and put to bed between flannelette blankets. The rectal temperature, pulse rate and respiration rate are recorded and a bed bath is given. Those with elevation of temperature are given a soft diet with extra fluids. Those whose temperature is normal receive ordinary diet with added nourishment, such as fruit juices. All children receive cod liver oil and tomato juice, as well as a mixture containing iron. Within the first few days of admission of a patient various tests are made, such as urinalysis, culture of organisms from the throat, Wassermann test, estimation of the sedimentation rate, blood count, Mantoux, Schick and Dick tests, electrocardiography and an X ray examination. If the patient suffers from chorea, the calcium, phosphorus and total protein content of the spinal fluid are determined.

As the children commonly remain in the ward for three months or more, there are special nursing problems. The little patients are taught to be passive and to have physical, emotional and mental rest. They are kept happy, warm and well fed. Bacal and Struthers observed that, after the acute infection had begun to subside, emotional outbursts became less frequent and coincidently the cardiac condition improved. Their investigations led them to believe that the sedimentation rate was the best criterion for determining the activity of rheumatic infection in childhood. They found that in acute rheumatic fever the sedimentation rate fell rapidly from a high level to a normal one following the subsidence of symptoms. They further ascertained that in chorea the sedimentation rate was constantly normal, or even below normal, in the absence of complications. Carditis was a manifestation in which, with recovery, the sedimentation rate fell slowly, over a period of months, to a normal level. The investigators also direct attention to their observation that rheumatic nodules tend to appear during the subsidence of the disease; that is to say, as the sedimentation rate falls. They noted also that a critical fall in the sedimentation rate might precede cardiac decompensation and the appearance of œdema, and was therefore of serious prognostic import.

When the time is opportune for graduated exercises, the patients are referred to the physical therapy department. They are graded into five classes. The first consists of those afflicted with organic heart disease, but able to carry on ordinary physical activity without discomfort. The second class embraces those suffering from heart disease and unable to perform ordinary physical activity without discomfort. The next class includes those with symptoms of heart failure when they are at rest. In the fourth class there are abnormal signs or symptoms referable to the heart, but the diagnosis of heart disease is uncertain. The final class embraces those with potential heart disease. In these there is as yet no definite circulatory disease, but it is advisable to follow them because the presence of an ætiological factor may cause heart lesions at a future date. Bacal and Struthers give an exhaustive account of the measures adopted by them in the rheumatism pavilion. Their communication is of considerable interest. Incidentally it might be mentioned that they include the controversial *erythema nodosum* amongst the rheumatic manifestations.

THE INFLUENCE OF CLIMATE ON METABOLISM.

B. Ahmad, R. B. Lal and N. C. Roy, in their report of a recent study of the basal metabolism of persons under varying conditions of temperature and humidity in India, remark: "The health status of an individual must necessarily be considered in terms of the range of adjustability which he possesses in relation to environmental variations."[1] This is true. Man probably is capable of greater adaptability to climatic changes than any other animal; but such changes must have an influence. Ahmad, Lal and Roy observed the effects on the basal metabolism of healthy subjects of change from the atmosphere of an ordinary room to a room cooled by air-conditioning. They found that the rate of basal metabolism decreased. They suggest that this decrease might be temporary only, that it might be followed by an increase "to balance the greater heat loss in temperate climates". The series investigated by Ahmad, Lal and Roy was too small to permit reasoned conclusions; but their method of investigation is interesting and might well be followed up in other parts of the world.

[1] *The Indian Journal of Medical Research*, July, 1938.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Amylase of the Subretinal Fluid.

H. J. M. Weve and F. P. Fischer (*Archiv für Augenheilkunde*, Volume CX, 1937) consider that amylase and peroxydase form a complex in the subretinal fluid. Under the same conditions these two substances diffuse with the same speed, and the size of the molecules of each is the same. Ascorbic acid (vitamin *C*) was the only substance found to have an inhibiting influence on the oxidizing effect of amylase.

The Proteolytic Ferments in the Subretinal Fluid.

H. J. M. Weve and F. P. Fischer (*Archiv für Augenheilkunde*, Volume CX, 1937) have shown that the proteolytic ferments in the subretinal fluid break down fibrin as far as the peptones and higher polypeptides. They are therefore to be classed as proteinases. The ferments probably come from the vitreous humour. Under physiological conditions the retina protects fibrin from the ferments of the vitreous. The lipoid content of the retina appears to give it this power. The authors quote the results of treatment of three patients with detachment of the retina to show the influence that these ferments may have. One detachment due to a typical dialysis, and therefore of excellent prognosis, could not be repaired by diathermy. The subretinal fluid was highly proteolytic. Another detachment, with very large superior tears, in an elderly patient, was repaired. The subretinal fluid was not very highly proteolytic. The subretinal fluid from the third patient was highly proteolytic. The tear recurred after diathermy, and then proteolysis had increased. Even though this detachment did not present any difficulties from the point of view of surgical treatment, it was repaired only after the fifth operation. Vitreous humour of animals and also that obtained in five cases in which no detachment was present, broke down when only small quantities of proteinase were present.

Inflammatory Pseudo-Tumours of the Orbit.

Hans Sauter (*Klinische Monatsblätter für Augenheilkunde*, Volume C, 1938) reviews Birch-Hirschfeld's classification of inflammatory pseudo-tumours of the orbit into three groups. The first type clinically resembles benign or malignant neoplasms, but is cured by medical treatment. The second group consists of those in which a tumour of the orbit appears to be obvious, but in which operation reveals nothing more than a cellular infiltration of the orbital tissues and extensive endarteritis without the formation of a tumour. Inflammatory pseudo-tumours of the third type produce a diffuse infiltration of the orbital tissues with lymphoid cells and characteristic follicular foci. These have been given the name of diffuse lymphomatosis of the orbit. The author describes two cases belonging to the second group. The first patient was a woman, aged fifty-four years, who complained that her right eye had been inflamed for five weeks and for several weeks had become progressively more prominent. Examination revealed œdema and swelling of the lids, an enlarged lachrymal gland, restricted eye movements and proptosis. Causes of disease were excluded. At operation no tumour was found. A portion of the orbital contents was removed and examined microscopically and was found to contain lymphocytes, other leucocytes and plasma cells in fatty tissue infiltrated with much connective tissue. The second patient, a woman, aged twenty-two years, had noticed prominence of one eye of one year's duration, with swelling of the upper lid. Examination revealed slight exophthalmos and a soft tender mass in the upper and inner part of the orbit. Biopsy revealed similar appearances to those found in the tissue removed from the first patient. Birch-Hirschfeld's classification has simplified the study of orbital pseudo-tumours. The name is applied to those tumours in which the ætiology is unknown, and therefore leuchæmic and pseudo-leuchæmic lymphomata and tuberculous and syphilitic granulomata are excluded. The author is convinced that the condition is rare; he has met with only two such cases in about fifteen years. He critically analyses the reports on 69 pseudo-tumours and believes that only seven can be considered as genuine inflammatory pseudo-tumours. The remainder, he maintains, should be classified as inflammatory exophthalmos. He suggests that if histological examination and inoculation of culture media and of laboratory animals had been carried out, their cause would have been found.

Ear Cartilage Graft for Spastic Entropion following Tarsectomy.

N. I. Shimkin (*The British Journal of Ophthalmology*, May, 1938) describes the operation of grafting a piece of cartilage from the ear to correct an entropion, which sometimes follows total tarsectomy. The pinna of the ear is bent forwards and an incision two centimetres long is made in the skin of the middle of the concha, the cartilage being exposed. The border of the flap is incised with a knife, the incision being two centimetres in length and 0·75 millimetre in depth. A strip 1·5 centimetres broad is sliced off with a knife held flat. An incision of the lid is made four to five millimetres from its border, and the skin separated up and down. The narrow strip of tarsus remaining near the cilia should be exposed. Three Snellen's loop sutures are used. The loops lie over the top of the cartilage. The needle passes in and out between the eyelashes. The sutures are tied and the three ends are drawn up and fixed with plaster above the eyebrow. The sutures are removed on the fifth day.

Iridencleisis.

J. Zipokes (*Archives of Ophthalmology*, April, 1938) describes a modification of iridencleisis to minimize manipulation of the iris and to conserve the epithelium. After the iris is grafted at the papillary border and drawn out of the wound, two meridional cuts are made, one on each side of the forceps. The resulting oblong-shaped flap of iris can then be easily laid out on the sclera like an apron, pigment side up.

The Use of Contact Glass for Cosmetic Purpose.

S. K. Mukerjee (*The British Journal of Ophthalmology*, January, 1938) describes the ocular condition of a girl, aged sixteen years, who suffered disfigurement from a leucomatous cornea, the result of smallpox. Several unsuccessful attempts had been made to darken the cornea by tattooing. A contact glass painted inside to match the other eye was inserted with excellent result.

Experimental Perforation of the Globe and its Relation to Treatment of Detachment of the Retina.

L. Weekers (*Archives d'Ophthalmologie*, March, 1938) demonstrates the important part played by the episcleral connective tissue in the healing of penetrating scleral wounds of the globe. It produces firm adhesion of chorioid and retina to the wound. Weekers's experiments were made on the eyes of guinea-pigs whose retinæ were detached in the course of preparation. He punctured the wall of the eye with a fine cataract knife or needle and examined the healed wound microscopically after a few days. He found that the chorioid participated slightly and the sclera not at all in the formation of the cicatricial plug that closed the puncture. The plug was formed almost entirely by proliferated episcleral connective tissue, which penetrated to the bottom of the wound, infiltrated the subjacent retina and even extended into the vitreous chamber, and thus formed a strong adhesion attaching the retina to the sclera. A certain amount of proliferation of chorioidal connective tissue supplemented the effect of the episcleral plug. These results were confirmed by similar experiments made on human eyes which had been enucleated. When a weak diathermy current is passed along the penetrating instrument after penetration,

a more pronounced chorioidal reaction is obtained, which supplements the major effect of the proliferation of episcleral tissue, and any small hæmorrhage is stopped by coagulation. Electrolysis may be used in place of diathermy. When perforation was made by the galvano-cautery, the tissues necrosed by the cautery formed an obstacle to the entry of the episcleral tissue, and the chorioiditis, though more extensive, did not form so firm a union. The chorioid also might become detached in course of preparation of the specimens; but if there was an episcleral link, as in the previous experiments with a cataract knife or needle, it always remained fixed.

OTO-RHINO-LARYNGOLOGY.

Nasal Sinusitis and Mental Disorder.

ALEXANDER BROWNLIE SMITH AND CHARLES MACDONALD ROSS (*Edinburgh Medical Journal*, May, 1938) examined for nasal sinusitis 808 patients suffering from mental disorder in the Royal Edinburgh Hospital. The methods used in this investigation were similar to those employed in the diagnosis of sinus infection in the ear, nose and throat department of the Edinburgh Royal Infirmary. A definite diagnosis rested on X ray evidence or on the presence of mucopus or pus in the material obtained by lavage from a suspected sinus. They conclude that approximately 12% of the patients in a large mental hospital suffer from nasal sinusitis. There is no evidence that nasal sinus infection plays any fundamental part in the causation of the vast majority of cases of mental disorder.

Treatment of Chronic Catarrhal Deafness with the Eustachian Heat Bougie.

EMANUEL SIMON (*Archives of Otolaryngology*, May, 1938) discusses the treatment of chronic catarrhal deafness with the Eustachian heat bougie. The Eustachian heat bougie is used with the ordinary Eustachian catheter. The bougie consists internally of two separate well-insulated circuits. The author concludes that the heat bougie will open the Eustachian tube for longer intervals than any other procedure. Opening up the Eustachian tube helps only those patients who have no scar tissue in the middle ear restricting movement of the ossicular chain. Chronic hyperplastic sinusitis is a highly prevalent disease. Chronic non-purulent *otitis media* is very common and is probably dependent for its source on chronic nasal infection or on chronic sinus infection, with its occasional acute flare-ups. These inflammatory processes are present in varying degree in most adults. Those affected may exhibit no symptoms, slight symptoms or pronounced symptoms. Chronic so-called catarrhal deafness may be diminished by improvement of the rhino-sinal mucosa and by direct treatment of the Eustachian tube. Unfortunately there is at present no known specific way to cure chronic sinusitis. By various empirical methods the condition of the mucosa in the nose and in the sinus may be improved to some degree, in most cases for a limited time; the inflammatory process in the ear is thereby alleviated or temporarily stopped. Direct treatment of the Eustachian tube, particularly with the heat bougie, is of value in many cases of chronic catarrhal *otitis media*, but not in all, since it does not remove ossicular restriction. Aeration of the middle ear does help some patients, even if only temporarily; it seems to tide others over acute tubo-tympanic catarrh, leaving the ears apparently well thereafter. In some cases the condition becomes worse in spite of all treatment. This is because it is impossible to arrest the course of the hyperplastic sinusitis which is the source and cause of chronic progressive *otitis media*.

Facial Palsy of Otitic Origin.

ABRAM H. PERSKY (*Archives of Otolaryngology*, April, 1938) discusses facial palsy of otitic origin. He concludes that while facial paralysis may occur before operation in cases of either acute or chronic suppurative *otitis media*, it is of greater significance and graver portent, both as to cure and as to the possibility of intracranial complication, in the chronic cases, especially in the presence of cholesteatoma. Hence, while the surgeon may be tempted to temporize when the disorder is acute, the situation is urgent in cases of chronic involvement. When the condition is simple and acute, a simple mastoidectomy should be the operation of choice. In the author's series the results obtained from complete radical mastoidectomy with exposure of the facial nerve and subsequent splitting of the sheath did not compare favourably with the results of simple mastoidectomy. But any facial palsy following operative procedure calls for an immediate exploratory operation, with an attempt to localize the site of injury and to expose the injured portion of the nerve. This is followed by simple drainage in cases in which there is a serous or a hæmorrhagic exudate, by removal of a spicule of bone, by splitting of the sheath, by immediate repair of the severed neurones, by approximation or by autoplastic graft from either proximal or distal nerves, according to the type of lesion found. Finally, the duration of the palsy may be an index of the prognosis. In cases in which the palsy is of short duration favourable results may be expected; but when the palsy has been present for a considerable period and reaction of degeneration has appeared, the prognosis is necessarily more guarded, the results depending on the success of repair or other surgical procedure performed on the nerve itself.

Thyroxin Therapy in Otosclerosis.

MAX A. GOLDSTEIN (*The Journal of Laryngology and Otology*, July, 1938) gives a report on forty-two patients suffering from otosclerosis who received thyroxin therapy. Twenty-three of the patients, or 54·8%, have shown definite improvement in hearing capacity, and in some cases cessation or reduction of tinnitus and a general clarification of speech perception have occurred. Ten patients, or 23·8%, showed slight improvement by comparison of audiograms made before and after injection; but this improvement was not stabilized according to the final audiograms six months later. In nine cases, or 21·4%, no improvement was noted after treatment. In no case did the author observe any decrease in hearing to follow these injections.

Progressive Facial Palsy Produced by Intratemporal Epidermoids.

GEOFFREY JEFFERSON AND A. ASHTON SMALLEY (*The Journal of Laryngology and Otology*, July, 1938) describe six cases of slowly produced facial paralysis associated with deafness. The palsies were due to latent, non-infected, petro-mastoid epidermoids. The authors believe that these epidermoids cannot, from their anatomical situation, be considered as secondary to epithelial invasion from without. The tympanic membranes were intact in all cases. A few isolated examples have been recorded previously, but the present group represents a clear clinical picture, now presented for the first time. It illustrates the pressure effects of epidermoids, in contrast with the better-known infective associations. Similar cases may remain undiagnosed because of the older, still dominant views on cholesteatoma formation. The nature of the epidermoids is discussed, and the view is held that most, if not all, of the present series were of embryonal origin.

Acute Pharyngeal Stenosis.

ROY SEELEY MOORE (*New York State Journal of Medicine*, March 15, 1938) discusses acute pharyngeal stenosis. He concludes that an acute stenosis of the pharynx only is occasionally met with, which will require either a tracheotomy, a tonsillotomy or a tonsillectomy. This condition occurs in association with abscesses in the region of the tonsillar aperture. An examination of this area should always be made before it is concluded that the closure is in the larynx. When the lesion is situated in the pharynx and drainage does not relieve the symptoms, it is much safer to remove all or part of the tonsils than to perform a tracheotomy.

British Medical Association News.

SCIENTIFIC.

A meeting of the New South Wales Branch of the British Medical Association was held on May 19, 1938, at the Royal Prince Alfred Hospital, Sydney. The meeting took the form of a number of clinical demonstrations by members of the honorary staff.

Carcinoma of the Ascending Colon.

Dr. B. T. Edye showed a male patient, aged fifty-two years, who had been admitted to hospital with a history of loss of weight and of pain and swelling in the right loin of three weeks' duration. There were no urinary symptoms. The bowels appeared to be normal. A large, firm, fixed, slightly tender swelling occupied the right loin and extended towards the right iliac region. The urine was normal, and excretion urograms revealed no impairment of renal structure or function. The leucocytes numbered 15,030 per cubic millimetre of blood, 79% being neutrophile cells.

On September 23, 1937, an incision was made in the right loin over the mass, and a large quantity of foul-smelling pus was evacuated. Bacteriological examination of the pus revealed actinomyces. Iodine was given by mouth, and a course of deep X ray therapy was prescribed.

On November 2, 1937, healing was complete and the patient left hospital. He was told to report at intervals, and it was proposed to have the colon investigated radiologically. However, several weeks passed before he returned, and he then complained of recurring attacks of colicky abdominal pain. Radiological examination after an opaque enema, on February 15, 1938, revealed an obstructive lesion of the ascending colon, strongly suggestive of carcinoma. On March 9, 1938, laparotomy was performed, a right paramedian incision being made. Carcinoma of the ascending colon adherent to the abdominal wall was discovered. It was decided to remove this in two stages. The first stage, which was carried out at once, consisted of a lateral anastomosis between the terminal portion of the ileum and the middle portion of the transverse colon.

On April 4, 1938, the right half of the colon was resected through an oblique right lateral incision. The tumour was adherent to the parietes, the involved portion of which was resected with the tumour. Recovery was uneventful. The pathological report stated that the growth was a columnar-celled adenocarcinoma of the ascending colon.

Osteomyelitis Simulating Osteogenic Sarcoma.

Dr. Edye's other two patients demonstrated the simulation of osteogenic sarcoma by osteomyelitis. The first was a male, aged thirty-two years. For five weeks prior to admission to hospital on April 5, 1938, he had pain in the upper third of the right thigh, ascribed to a slight injury. The pain was aching in character, persistent and worse at night. The movements of the hip joints were somewhat restricted on account of the pain. Night sweats had occurred on several occasions. The previous health had been satisfactory.

On examination tenderness and swelling were noted on the medial side of the right thigh at the junction of the upper and middle thirds, extending obliquely upwards towards the base of the great trochanter. The swelling was related to the bone. The overlying skin was normal in appearance. The movements of the right hip joint were restricted and the quadriceps muscle was wasted. No other symptoms were present. A blood count gave the following information: the erythrocytes numbered 5,090,000 per cubic millimetre, and the hæmoglobin value was 101%. The leucocytes numbered 6,125 per cubic millimetre; of these 78·5% were neutrophile cells, 12·5% were lymphocytes, 6% were monocytes, and 3% were eosinophile cells.

The first report of the radiographic appearances of the tumour was the following:

> A large osteogenic sarcoma involving upper end of shaft of the femur just below lesser trochanter; rapidly growing.

X ray examination of the chest revealed no abnormality. A course of deep X ray therapy was prescribed, and eight days after its completion the tumour was again examined radiologically. The report stated that considerable resolution had taken place since the previous examination. The patient was prepared for operation, suitable blood donors were typed, and the director of the department of pathology of the hospital was to be present in the operating theatre to report upon frozen sections of tumour tissue excised for the purpose.

At the operation a vertical incision was made over the most prominent part of the swelling. As the bone was approached, dense whitish tissue was cut and portion was excised for biopsy. This proved to be of an inflammatory nature. The incision was deepened and extended, and a cavity containing pus was opened. The pus contained *Staphylococcus aureus*. The cavity extended into the shaft of the bone, and it contained several sequestra. There was evidence of new bone formation in the overlying tissues. The cavity was dried out, the sequestra were removed and overhanging margins of shaft were cut away. The whole cavity was then filled with vaseline and gauze.

Dr. Edye's last patient was a male, aged thirty-six years. He was first admitted to hospital on July 6, 1937, with osteomyelitis of the right side of the skull complicating a carbuncle in the right occipital region. Drowsiness, mental instability, vomiting and weakness of the left arm and leg were present.

The scalp was incised and much pus was evacuated. An extensive area of the skull was bare and eroded, but a trephine opening revealed no dural pus. The infecting organism was the *Staphylococcus aureus*, and blood cultures yielded a growth of this organism over a period of ten weeks. Later a large right cervical abscess and one over the angle of the mandible on the right side were incised.

For many weeks the patient's life was in jeopardy, but he began to improve slowly, and finally recovered, apparently completely.

On March 9, 1938, he was readmitted to hospital. He complained of an aching pain in the middle of the right thigh. The pain had begun gradually three weeks previously and was worse at night. Examination revealed deep swelling and tenderness along the lateral aspect of the middle third of the right femur. The patient's temperature was 37·3° C. (99° F.) and his pulse rate was 125 beats per minute. X ray examination revealed extensive necrosis of the posterior aspect of the femur. Radiologically it was not possible to differentiate between osteogenic sarcoma and osteomyelitis.

At operation pus was excavated, and an irregular cavity was found in the middle third of the femoral shaft from which sequestra were removed. *Staphylococcus aureus* was grown on culture from the pus. The pathology of the condition was that of osteomyelitis of gradual onset.

Old Fracture-Dislocation of the Lumbar Spine with Compression of Nerve Roots.

Dr. R. A. Money, in collaboration with Dr. E. P. Susman, showed a male patient, aged fifty-five years, who had sustained a fracture-dislocation of the fourth and fifth lumbar vertebræ nine years earlier. The deformity had not been reduced. X ray films of the deformity were shown. Since that time the patient had had severe pain in both legs, and weakness and wasting in his right thigh and leg. He could only hobble around with a crutch. The backward dislocation of the fifth lumbar vertebra on the fourth had been causing pressure on the *cauda equina* and on the fourth and fifth lumbar nerve roots at the intervertebral foramina, especially on the right side.

An attempt at reduction by forcible manipulation under spinal anæsthesia was unsuccessful. Laminectomy was therefore performed, and the spines and laminæ of the fourth and fifth lumbar vertebræ were removed. An old hæmatoma with chocolate-coloured fluid was found intradurally on the right side. The nerve roots on both sides were found to be compressed between the laminæ and pedicles of the dislocated vertebræ. They were completely freed into their intervertebral foramina.

Convalescence was uneventful. Relief from the pain in the legs was obtained almost immediately. Muscle tone and power returned slowly, and when the patient left hospital six weeks later he walked comfortably with the aid of a stick.

Meningioma of the Orbital Roof.

Dr. Money and Dr. Susman next showed a female patient, aged thirty-seven years, who had been "nervy" for eighteen months. She had been noticeably growing duller mentally, unable to concentrate and to carry on her work as a telephonist. Recently, headaches had been severe, and her eyesight was failing. Character changes had also become very pronounced.

On examination she was euphoric and made witty remarks the whole time. Dr. Money remarked that this was a characteristic accompaniment of frontal lobe lesions, called *Witzelsucht*. Double papillœdema was present. The visual fields and sense of smell could not be tested accurately owing to lack of cooperation; otherwise, no abnormality was found on neurological examination. Radiological examination revealed a characteristic thickening of the right orbital plate of the frontal bone. Lumbar puncture yielded yellowish-brown fluid, the protein content being 280 milligrammes per 100 cubic millimetres.

Operation was undertaken in two stages.

At the first operation ventriculography was performed. Dr. Money showed films in which displacement to the left of the anterior horns of both ventricles could be seen. The right anterior horn was small and displaced far backwards. A scalp flap was reflected forwards from a transverse coronal incision and then a right frontal bone flap was raised. A large meningioma was seen growing from the roof of the right orbit and displacing the frontal lobe upwards and backwards. The intradural tension was so great it was impossible fully to close the dura; the bone flap had to be sacrificed, and only the scalp tissues were replaced.

At the second operation, a week later, the tumour was removed and its origin was destroyed by diathermy after partial amputation of right frontal lobe had been performed. Convalescence was delayed by a mild wound infection and by a discharging sinus, which had healed. At the time of the meeting the patient's general and mental condition was normal. The papillœdema had completely subsided. The right frontal area was rather unsightly and pulsated with the brain. Dr. Money said that in a year's time, if the patient remained well, the bone flap would be replaced by a graft. The pathological examination of the tumour showed it to be a typical meningioma.

Parasagittal Meningioma.

Dr. Money then showed a female patient, aged forty-four years, who complained of fits over a period of ten years. She would experience an aura of flexion in her right arm and leg, and then become unconscious for several minutes. She often had minor paroxysms accompanied by flexion of the arm and leg, without losing consciousness. Occasional headaches had recently occurred. In between fits she was well.

On examination her cranial nerves were intact and her fundi were normal. The visual fields were full. There was some motor weakness of the right arm and leg, and the reflexes on the right side were exaggerated. Ankle clonus was elicited on the right side, and the right plantar reflex was extensor in type. Sensation was not interfered with. Lumbar puncture was performed and clear fluid was obtained at a pressure of 150 millimetres of water. The protein content was not increased.

By ventriculography, the right lateral ventricle and the third ventricle were seen to be displaced to the right. The body of the left lateral ventricle appeared to be compressed from above and deformed. The temporal horn of the left ventricle appeared on the films to be somewhat distorted.

At operation a left fronto-parietal bone flap was turned down. A large tumour was found growing from the *falx cerebri* and compressing the medial part of the left cerebral hemisphere, in the region of the Rolandic area. The tumour was wholly removed, and its origin was destroyed by diathermy. The tumour was exhibited, as well as a microscopic slide which showed it to be an angioblastic meningioma.

After operation the patient had aphasia and complete right-sided hemiplegia for about ten days, but recovery had since taken place except in the right lower limb, which was spastic and paretic at the knee and ankle joints at the time of the meeting.

Craniopharyngioma: Suprasellar Cyst.

Dr. Money's next patient, a boy, aged six years, had first been seen by Dr. Money three years earlier, when he had complained of recurrent attacks of headache, vomiting and failing vision, together with dizziness and unsteadiness on his feet. Nothing definitely abnormal was detected at that time by ventriculography. He was regarded as suffering from posterior basal meningitis, and was kept under observation.

A year before the meeting polyuria and polydipsia developed, with periods of somnolence. Bitemporal hemianopia was detected on the Bjerrum screen. Evidence of increased intracranial tension was seen in the ordinary skiagrams, but no actual suprasellar calcification was visible.

At operation, a right frontal flap was reflected, and a large craniopharyngeal cyst was found by needling through the right frontal lobe. The cyst contained about 60 cubic centimetres (two ounces) of typical chocolate-coloured fluid, full of cholesterol. Drainage had been repeated through one of the drill holes every two to three months since the operation, whenever the pressure in the cyst became high. Dr. Money said that recently, after drainage, the cyst cavity had been filled with air, and special X ray films had been taken in four positions. These showed, in a remarkable way, the extent of the cyst and the solid portion of the growth. At the time of the meeting the patient was well. He was symptom-free, and had full visual fields between tappings.

Bone Graft for Traumatic Hernia Cerebri.

The next patient shown by Dr. Money was a boy, aged nine years, who had been knocked down by a taxi-cab six months earlier and had sustained a compound comminuted depressed fracture of the skull in the right temporal region, with lacerations of the dura, from which the brain was protruding. Consciousness had not been lost. At another hospital the depressed bone fragments and foreign bodies had been removed, and after *débridement* the scalp wounds had been sutured. Infection occurred, and the sutures had to be removed. This caused the formation of a *hernia cerebri*. The wound discharged for many weeks and finally healed, a pulsating swelling being left. Otherwise the child was well and had no symptoms.

When he was examined early in April, 1938, a meningo-encephalocœle was visible and palpable in the right temporal region. The upper part pulsated and could be reduced with change of posture; the lower part could not be made to disappear even with pressure. Dr. Money exhibited photographs of the condition and plain skiagrams. These showed the swelling, and an area of missing bone and old fracture in the right frontal and squamous temporal bones. Encephalograms, revealing a diverticulum of the anterior horn of the right lateral ventricle, drawn out into the hernia, were also shown.

At operation, a frontal scalp flap was reflected forward from a transverse coronal incision, and the edges of the gap in the bone were defined. A mass of necrotic brain and fibrous tissue was found protruding through the deficiency in the bone. This was apparently continuous with the dura and cortex. It appeared to have been the site of an old abscess. This was freed and amputated. The outer table of portion of the neighbouring bone was then chiselled off and inserted as a graft to fill the gap, and the frontal scalp flap was replaced over it.

The wound healed by first intention, and at the time of the meeting no pulsations or swelling could be seen or felt.

Multiple Fractures of the Face and Jaws.

Dr. Money then showed a girl, aged seventeen years, who had been severely injured in a motor car accident. She had not been rendered unconscious, but she received many

lacerations of the nose and face, with multiple fractures of the facial bones but without fracture of the skull. The most important damage occurred to the mandible, which was fractured on the left side behind the last molar tooth, with much displacement. The whole region was terribly swollen and there was much nasal discharge. The occlusion of the patient's incisor teeth had never been satisfactory, but it was now worse. As no teeth were available in the posterior fragment of the mandible for wiring, open operation had to be resorted to. An incision was made in the skin crease under the mandible; the fragments were isolated without entry into the buccal cavity, and two pieces of silver wire were inserted to hold them together. The result obtained was shown in X ray films with the wires *in situ*. The mouth could be opened to its full extent and mastication was satisfactory.

Lung Abscess Followed by Pulmonary Tuberculosis: Healed by Artificial Pneumothorax.

Dr. Cotter Harvey showed a female patient, aged twenty-eight years, who had presented on November 14, 1936, with a history of cough and sputum of nine months' duration following the extraction of twenty-four teeth under a general anæsthetic. She had had several short periods of rest in bed, with amelioration of symptoms; but during the previous month her sputum had increased in amount to three-quarters of a cupful daily, and had become foul in taste. She had had several slight hæmoptyses.

On examination she was seen to be a pale woman, weighing 39·5 kilograms (six stone three pounds). This represented a loss of 8·2 kilograms (eighteen pounds) in nine months. Slight clubbing of the fingers was noted. The percussion note was impaired over the right lower lobe, where the breath sounds were diminished, and râles were heard below the right scapula. Radiological examination revealed a large area of consolidation, chiefly in the right middle zone, with a central cavity, which the radiologist regarded as being probably a lung abscess, possibly tuberculous.

The leucocytes numbered 22,200 per cubic millimetre of blood and the red blood cell sedimentation rate was greatly increased. No tubercle bacilli were found in the sputum at three examinations.

With postural drainage and rest in bed, the patient made steady improvement. She was discharged from hospital two months later, with a slight cough and no sputum. Radiological examination at that time indicated fibrosis and apparent healing of the condition.

In October, 1937, she was seen again. After six months without symptoms, her cough had returned. She was voiding each day about a quarter of a cup of sputum. She had had no further hæmoptyses. There were now signs of a large cavity in the right lower lobe. X ray examination confirmed this and revealed a much larger cavity than had been present nine months previously. Tubercle bacilli were found in her sputum on three successive examinations.

Artificial pneumothorax was instituted and good collapse was obtained, especially of the lower lobe. By February, 1938, her cough and sputum had disappeared and she was steadily gaining weight. She was having regular fills of about 900 cubic centimetres at three-weekly intervals. She had become quite free from symptoms. The last skiagram, made on May 11, indicated that the cavity had completely filled in. Dr. Harvey said that he proposed to continue the treatment for at least a further two years. He considered it probable that a tuberculous lesion had become engrafted on an area of lung damaged by chronic sepsis, but he admitted that it could not be denied that the process might have been a tuberculous one from the beginning, with apparent quiescence and subsequent relapse.

Cystic Disease of the Lung.

Dr. Harvey also showed a patient and a number of skiagrams of other patients suffering from cystic disease of the lung, to illustrate various complications and errors in diagnosis. He regarded the condition as not uncommon if astute radiological observation was made and if the possibility of its presence was kept constantly in mind.

Radical Mastoidectomy.

Dr. G. C. Halliday showed a male patient, aged twenty-two years, who had suffered from a chronic foul discharge from his left ear for eight years, with cholesteatoma coming down from an attic perforation. His hearing before operation was practically nil. A radical mastoidectomy was performed, with the technique as devised and adopted by Dr. R. S. Godsall. In this operation no flap was cut from the posterior meatal wall. The result aimed at was to leave the external meatus, attic and middle ear as one canal, and obliterate the mastoid antrum by scar tissue.

Peripheral Neuritis.

Dr. A. S. Walker showed a Maltese woman, aged thirty-three years, who had had pains in the legs for a period of five months and who had been unable to walk for over four months. She had felt "pins and needles" in the legs for over two months before her admission to hospital, and within a few weeks of the onset was unable to walk on account of loss of power. At the same time altered sensation and tingling were felt in the fingers, and she noticed that her hands were weak and clumsy.

Examination of the nervous system revealed no affection of the cranial nerves, but extensive wasting of the limb muscles, most intense in those of the lower part of the leg and the forearm. These muscles were very tender. The intrinsic muscles of the hands were also very wasted. There was delayed conduction of tactile and thermal sense over the glove and stocking areas. The plantar reflexes were flexor in type, and knee jerks and ankle jerks were absent.

The cerebro-spinal fluid was under a pressure of 110 millimetres of water. There was no increase in the cell content. The chloride content was 720 milligrammes per 100 cubic centimetres, the sugar content was 60 milligrammes per 100 cubic centimetres, the protein content was 20 milligrammes per 100 cubic centimetres. The gold sol test elicited the figures 0011211000. There was no reaction to the Wassermann test. The systolic blood pressure was 135 and the diastolic 65 millimetres of mercury. A blood count revealed 5,280,000 erythrocytes and 6,950 leucocytes per cubic millimetre. The hæmoglobin value was 95%. Of the leucocytes 60·5% were neutrophile cells. There was no excess of stippled cells. No arsenic or lead was found in the urine, hair, nails or skin of the feet.

On the patient's admission to hospital a dermatitis of the exfoliative type was present on the lower limbs.

Dr. Walker remarked that the ætiology was of interest. There was no positive evidence of arsenical poisoning, but some remarks made by the patient, whose restricted vocabulary was a drawback in the taking of an accurate history, suggested that some such risk might have been incurred. Further specimens of nails and hair had been submitted to analysis. If the condition was not due to a chemical cause it might be of infective origin, polyneuritis of virus type. Recovery, as might have been expected in view of the extensive wasting, was very slow. A thorough trial of a preparation of vitamin *B* suitable for injection had been tried. The clinical impression gained was that a more rapid amelioration of pain and discomfort took place after the injections. Discussion with other members on this case elicited a general consensus of opinion that arsenic was the most likely cause. It was agreed that further chemical investigation was desirable.

Aortic Regurgitation.

Dr. Walker's second patient was a female, aged fifty-four years, who had complained of breathlessness only for four weeks before her admission to hospital a month earlier. For four weeks she had felt breathless on exertion; and just before admission to hospital her attacks of breathlessness had become more severe, lasting up to twenty minutes, and had also occurred at night after she had gone to bed, independently of exertion. The only illness in her past history was typhoid fever in 1916. She had no luetic history.

Examination of the heart revealed gross enlargement to the left, with a heaving, diffuse, apical impulse. There was a classical water-hammer pulse. The systolic blood pressure was 220 and the diastolic 60 millimetres of mercury. A loud to-and-fro murmur was audible in the great vessel area, propagated up into the neck and to the apex of the heart. At the time of the patient's admission to hospital moist sounds could be heard at the base of each lung. The reaction of the pupils to light was absent on the right side and sluggish on the left. The knee jerks were absent on the right side, and the plantar reflexes were flexor in type.

The blood yielded a very strong reaction to both the Wassermann and Kline tests. Only a slight grade of anæmia was present, the erythrocytes numbering 4,160,000 per cubic millimetre and the hæmoglobin value being 71%. X ray examination of the heart revealed pronounced enlargement, especially of the left ventricle. There was also evidence of pulmonary congestion. There was a slight fusiform enlargement of the aortic arch, but no aneurysm.

Dr. Walker remarked that the case was a typical example of specific aortitis. The patient was shown to illustrate the remarkably short history which such patients sometimes gave. It was noteworthy that the condition should remain latent until she reached the age of fifty-four years and then rapidly produce a striking nocturnal dyspnœa. She had improved greatly with rest and general treatment, and she was having mercury and iodides in addition.

Spinal Neoplasm.

Dr. Walker's third patient, a female, aged fifty-seven years, had been admitted to hospital complaining of weakness of the legs and pains in the knees and lower part of the legs for the preceding three or four months. She first noticed staggering in her gait, and the right leg appeared to get in front of the left when she walked. The feet became cold and numb, with some tingling sensation up to the middle of the thighs, and she suffered from spasmodic attacks of cramp. During the previous three months she had been a little dizzy and breathless, with intermittent headaches. Latterly there had been some incontinence of urine and fæces.

Physical examination revealed that the cranial nerves were intact, but gross lack of coordination in the lower limbs, with some impairment of motor power. The latter, however, was less evident than the lack of coordination. The senses of touch and pain were grossly impaired in both legs, but the temperature sense was satisfactory. Both plantar reflexes were extensor in type, and all the lower limb deep reflexes were greatly increased. Ankle clonus was present on both sides. No definite abnormality was found on general examination. The systolic blood pressure was 145 and the diastolic 85 millimetres of mercury. A blood count revealed 5,350,000 erythrocytes and 8,350 leucocytes per cubic millimetre. The hæmoglobin value was 103% and the colour index was 0·9. Neither the blood nor the cerebro-spinal fluid reacted to the Wassermann test. The cerebro-spinal fluid flowed at a pressure of 120 millimetres of water. There was a positive response to the Queckenstedt test. The cerebro-spinal fluid was yellow and contained 1,000 milligrammes of protein and 710 milligrammes of chloride per 100 cubic millimetres. Coagulum readily formed. No lipiodol examination had been made. Dr. Walker remarked that one of the chief points of interest was the unusual degree of loss of deep sensibility in the lower limbs, which at first aroused a suspicion of subacute combined degeneration of the spinal cord.

Probable Streptococcal Infection with Pneumonia and Pericarditis Treated with Sulphanilamide.

Dr. Walker's next patient was a male, aged twenty-seven years, who was shown to illustrate recovery after severe pneumonia with pericarditis probably of streptococcal origin. He had a history of three attacks of pneumonia nine or ten years earlier, and in 1936 he had pleurisy. Radiographic examination of the chest at this time had revealed evidence of chronic bronchitis but not of tuberculosis. Early in 1938 he had an influenzal infection, after which a post-nasal discharge was troublesome. This was due to antritis; and on February 28, 1938, double radical antrostomy was carried out. Two weeks later he had swelling of the joints, particularly the hands, but this cleared up in twenty-four hours. Two weeks later again, after some irregular pyrexia, he had pain in the chest, and complained of a sense of compression beneath the sternum, with pain on breathing. Signs of pneumonia at the base of the left lung were found, and also of pericarditis. In the early stage of this condition there were 20,000 leucocytes in a cubic millimetre of blood, but on his admission to hospital on March 27, 1938, the leucocyte count had dropped to 8,100. There was pronounced pericardial friction in the fifth left interspace 2·5 centimetres (one inch) from the sternum over a moderate area. The sputum contained no tubercle bacilli, and streptococci predominated.

Attempts at culture from the blood were without result. X ray examination revealed pleural thickening at the left base and a general increase in lung markings. The question of sulphanilamide therapy was approached with caution on account of the falling leucocyte count. The drug was, however, given by mouth, two tablets being taken three times a day, and a daily leucocyte count was made for some days. The clinical condition, which had occasioned great anxiety, improved, and the leucocytes rose to 9,050, and then to 14,750. A week later the leucocytes numbered 12,675. In all these examinations the proportion of polymorphonuclear cells was 70% to 80%.

Dr. Walker remarked that the evidence seemed to point to a systemic streptococcal infection causing joint swelling, pneumonia and pericarditis. It was of interest that sulphanilamide appeared to exercise a very favourable influence on the condition without any toxic effects, in spite of the threatening fall in the number of leucocytes. His progress had been interrupted by some setbacks in which the signs in the left lung flared up again. This condition was settling down well, however, and his cardiac condition appeared to be quite satisfactory.

"Cardiazol" in Psychoses.

In presenting patients to illustrate the use of "Cardiazol" in psychoses, PROFESSOR W. S. DAWSON explained that convulsion therapy had been introduced by L. von Meduna, of Budapest, in 1934. He had acted on the suggestion that there was a biological antagonism between epilepsy and schizophrenia, because of the rare association of the two and the disappearance of schizophrenic symptoms after convulsions. After experiments on animals Meduna induced fits first of all in man by intramuscular injections of camphor in oil, then by "Cardiazol" given intravenously in aqueous solution. Professor Dawson said that in the present series a 10% solution had been used, buffered to pH 8·0 by the addition of di-sodium hydrogen phosphate. There was said to be less tendency to thrombosis and fibrosis of the injected veins when this was done. The injection should be given after several hours' fast. According to Meduna's last published paper, the first injection should be of five cubic centimetres of a 10% solution. If this failed to produce a convulsion another six cubic centimetres should be given immediately. Should this additional amount also fail, another attempt should be made next day with seven cubic centimetres, with an additional eight cubic centimetres if necessary, and on the following day with nine and a further ten cubic centimetres should no convulsion be produced. In the treatment of patients at the meeting it had not been necessary to exceed a single dose of eight cubic centimetres. Meduna reported that in one case he failed to produce a convulsion until the fourth day, when he had to give eleven cubic centimetres, followed immediately by another twelve cubic centimetres. He stated that he had seen no harm result from three injections of "Cardiazol", 16 cubic centimetres, 16 cubic centimetres and 14 cubic centimetres, a total of 46 cubic centimetres (or 4·6 grammes) of "Cardiazol", given in three successive minutes. A successful intravenous injection, which had to be given as quickly as the piston of the syringe could conveniently be propelled, was followed

within from ten to thirty seconds by a cough, when a gag should be inserted. Then followed aimless or perhaps defensive movements of the arms, and next a general stiffening, in which the patient usually assumed the posture of extensor rigidity. This phase lasted from 10 to 25 seconds. After this the mouth was opened, when the gag should be adjusted or inserted, and clonus developed. It lasted for from 30 to 45 seconds, and was followed by coma in 5 to 10 minutes.

Meduna stated emphatically that the convulsions should be induced at least twice a week. The practice at the Royal Prince Alfred Hospital was to give this treatment three times a week. Meduna gave twenty fits as the minimum course, and advised that at least three more fits should be induced after a full remission of mental symptoms had occurred. Professor Dawson remarked that two patients in his own series seemed to have done perfectly well after eight fits. Meduna advised as many as thirty fits before hope of a favourable response was given up in a likely case. He also laid particular stress upon the importance of therapeutic talks given after at least every fifth convulsion, preferably on the day following the injection. Cardio-vascular disease was a contra-indication, and the treatment should be suspended during febrile or other intercurrent illnesses.

Professor Dawson went on to explain that the best results were obtained with patients treated within six months of the first manifestation of schizophrenic symptoms, for whom Meduna claimed a remission rate of 80% to 90%. No good could be anticipated from the treatment of patients having a history of schizophrenia of more than three years' duration. It seemed to be the general experience that stuporous and confused patients reacted more favourably than those whose chief symptoms were delusions. Elated and excited patients did not as a rule react favourably. Von Meduna stated that he got the best results in the treatment of katatonics and hebephrenics whose illness was of recent onset.

The first patient shown by Professor Dawson was a boy, aged sixteen years, an apprentice, who had been referred to him by Dr. E. Cook. The boy had been irritable since the beginning of the year. Three weeks before his admission to hospital he had become excited and had been taken by ambulance from his work to hospital. He was sent home after being kept under observation for a few hours. He displayed obsessions about time, counted his steps, seemed preoccupied and made constant reference to a sense of change. His conversation was mostly disconnected, but he complained that his mind was affected and expressed fears that there might be something wrong with his spine. He also stated that he was changing from a boy to a man in the course of a week.

On March 9, 1938, he was admitted to the psychiatry pavilion. He was fidgetty, preoccupied, dull and unable to express himself coherently. After three or four induced convulsions he became brighter and more alert. He received nine injections of from six to eight cubic centimetres, eight of which were followed by convulsions. He improved steadily, was discharged from hospital on April 11, 1938, and had been well since.

Professor Dawson remarked that it would perhaps have been fairer to term the boy's illness instability of adolescence rather than schizophrenia. The prognosis was by no means unfavourable from the beginning, but it seemed likely that the "Cardiazol" treatment accelerated recovery.

Professor Dawson next showed a female patient, aged twenty-five years, who had been engaged in domestic duties. She had been referred to him by Dr. G. F. L. Elliott. She was sensitive and retiring, with few companions. Three months earlier she had become depressed and had displayed antagonism towards other members of her family, and especially towards her younger sister who had just married and who, she declared, was suffering from tuberculosis. She had also displayed general loss of interest and disinclination for her usual activities.

She was admitted to the psychiatry pavilion on March 10, 1938, and received her first injection on March 16, 1938. She had in all eight good fits, induced at first at the rate of three a week, then one a week. By March 28, 1938, she was already brighter and had lost her suspicions and morbid antagonisms. She was discharged from hospital on April 12, 1938, feeling very well and appearing bright and interested. She had been well since.

The third patient shown by Professor Dawson was a girl, aged nineteen years, who had been referred to him by Dr. A. T. Chapple. She had lived quite an active life, swimming, dancing and playing tennis, and had also sustained successfully a major part in a play. About twelve months before the time of the meeting she had become moody and mildly depressed, and complained of inability to assert her personality. She stated that she felt oppressed by the presence of other people, and that the neighbours were trying to absorb her brain. She developed numerous suspicions and antagonisms. She had always been of a reserved temperament, but during the past year she had avoided her friends. The patient had good insight into many of her symptoms; she was keenly aware of feeling different and of failing in concentration and interest, but would not admit that her suspicions might lack foundation. Her conversation was often disconnected; for example, she complained that in one hospital the people on the upper floor moved the furniture about so that her kidneys, heart and other organs were displaced. She also stated that when she travelled on the ferry the men banged things down on the wharf and caused her heart to be shifted. She made numerous other odd references to the dominance of other people and to the loss of her own personality.

She was admitted to the psychiatry pavilion on March 16. Up to the time of the meeting she had had fourteen intravenous injections of "Cardiazol" followed by convulsions, and one injection which produced only a mild mental confusion. The doses varied between five and seven cubic centimetres. She had become brighter and was more alert and less self-absorbed, but was still inclined to be suspicious and to project her sense of inferiority. Professor Dawson remarked that her condition was hebephrenic in type, and improvement was only moderate. She would have to undergo a full course of at least twenty fits in order to give the treatment a proper trial.

The fourth patient shown by Professor Dawson was a labourer, aged thirty-two years, who had been referred to him by Dr. R. E. Wherrett. The patient was unmarried. His mother reported that six months earlier he seemed depressed and forgetful, was apt to repeat what he had said and seemed to lose interest easily. He had continued to work up to a more recent date, but finally felt unable to carry on. He then expressed fears of having contracted venereal disease and remorse for things he had done years earlier. He said that he was put under a test, that his thoughts were not his own and that he was talked about by strangers in the street. He seemed moderately depressed. His physical condition was satisfactory, but he had had ten teeth extracted recently, and several carious teeth remained. He was admitted to the psychiatric pavilion on April 3, 1938, and treatment was begun a few days later. He had had ten good reactions up to the time of the meeting; two injections had not produced convulsions, but only an unpleasant sense of anxiety and confusion. Up to five days before the meeting he was remarkably better and felt and looked bright. He then complained of malaise and had a slight pyrexia, with an occasional rise in pulse rate up to 130 beats per minute. He also became mentally unsettled and depressed, but was already feeling better both physically and mentally. Treatment had been suspended until the pyrexia had subsided.

Professor Dawson said that his illness was more melancholic in type and bore a good prognosis. Up to the development of the influenzal attack the patient was showing great improvement. He often reported that he felt very well during the afternoon after the fit, although earlier he had been inclined to relapse again the following morning. He had required doses of from seven to eight cubic centimetres of the 10% solution of "Cardiazol".

The last patient shown by Professor Dawson was a young woman, aged twenty-seven years, a secretary, who had been referred to him by Dr. A. S. Evans. Two weeks before her admission to hospital, on April 20, she had returned from work saying that her work-mates were looking at her and talking about her. After a day or two she returned to work, but a week later felt unable to continue. She was then depressed and spoke very little, was content to lie in bed and displayed no desire for any activity. Her bladder had to be catheterized on occasion and she also required spoon feeding.

She had lived a rather secluded life, and had few friends of either sex. She had been in a position as a private secretary for eleven years. Just before the onset of the psychosis she had had several teeth extracted, one of which had a cyst attached.

On admission to the psychiatric pavilion on May 3, 1938, she was depresed and even apathetic, replying "yes" and "no" to some questions. She said that she did not feel ill. Three days after her admission she seemed brighter, but relapsed again, and remained mute and depressed for another two weeks until the afternoon of May 19. She had had injections of "Cardiazol", six or seven cubic centimetres, on May 13, 16, and 18. Professor Dawson felt agreeably surprised to find this patient sitting up on the couch at the meeting, responding readily, looking bright, and stating that she felt much better. She explained that her previous inertia was just due to laziness. Professor Dawson remarked that the prognosis for this patient appeared to have been quite favourable. Nevertheless he felt that the "Cardiazol" treatment was responsible for an unexpectedly rapid improvement.

Meningioma.

DR. GILBERT PHILLIPS showed a male patient who had suffered from headaches since 1934. These had commenced as a supraorbital pain on the left side. He had felt "run-down" for two years. His memory and power of concentration had been poor for nine months. He had had difficulty in finding the right word when speaking for six months. His right leg had felt heavy and had been inclined to drag for a period of four months.

He had become unconscious during defæcation two months prior to the meeting. His sister noticed that he had been dragging the right leg for two months. He had six diopters of bilateral papilloedema, with recent hæmorrhages, particularly on the left side. He had right congruous homonymous hemianopia with macular sparing. Pronounced weakness of the right leg, with increased reflexes, was noticed. The cerebro-spinal fluid was under a pressure of 280 millimetres of water, with no increase in the protein content.

On March 31, 1938, a first-stage craniotomy was carried out. A large tumour was found, presenting at the occipital pole on the left side. The appearance at first resembled that of a glioma; but after removal of the supero-lateral portion of the occipital lobe the tumour proved to be a huge meningioma arising from the *torcular Herophili*. About thirty or forty grammes of tumour were removed by endothermy loop. Continuous blood transfusion was necessary. The bone flap was removed and autoclaved. The patient responded to questions at the conclusion of the operation.

On April 1, 1938, the patient's general condition was excellent, except that he had total right upper monoplegia.

On April 5, 1938, a second-stage craniotomy was performed. The flap was reflected and the remainder of the tumour was removed, with the exception of a small portion adherent to the junction of the longitudinal and transverse sinuses, which was treated by heavy endothermy coagulation. Necrotic brain tissue anterior to the tumour was sucked out.

On April 6, 1938, the patient's condition was excellent. Considerable improvement had already taken place in the power of the right arm.

Cystic Glioma.

Dr. Phillips next showed a specimen of cystic fluid to demonstrate the huge quantity of fluid which might accumulate slowly in an intracranial neoplasm. The patient had been operated on on June 5, 1936, and the case had been reported in THE MEDICAL JOURNAL OF AUSTRALIA of November 21, 1936, at page 714. At that time a subtotal removal had been made of a tumour in the right temporal lobe, which proved to be an oligodendroglioma. At that time the neoplasm did not contain any cystic fluid. The patient had been treated with massive doses of deep X rays by Dr. A. T. Nisbet and had been very well since operation until four or five months before the meeting. Three weeks prior to the meeting it was decided to insert a needle through one of the burr holes to determine whether the neoplasm had become more extensive. Cystic fluid was encountered, about 10 cubic centimetres being lost before an attempt was made to collect it. In the following ten minutes another 245 cubic centimetres of fluid (eight ounces) were removed from the cyst. The patient was in no way distressed by the procedure and carried out an ordinary conversation during the drainage. Dr. Phillips remarked that it seemed quite likely that this cystic change was the result of deep X ray therapy.

Buccal Epithelioma.

DR. H. J. HAM showed a male patient, aged forty-five years, who had been referred to him by Dr. T. M. Furber on November 4, 1937, with an extensive proliferative epithelioma of the mouth. The growth involved the right lower alveolus, from the region of the third molar tooth to the lateral incisor, and extended to the floor of the mouth as far as the mid-line. No involvement of the cervical glands could be detected by palpation. No response was elicited to the Wassermann test and X ray examination of the right mandible revealed no bone involvement.

The patient was first treated by deep X ray therapy, 2,000 *r* being given to each of two fields. One dose was given obliquely from below to the right submaxillary region, and the other laterally to the right jaw. This produced a reaction of fibrinous desquamation over the region of the growth. When the patient was seen one month later the reaction had subsided and the growth was smaller.

Two months after the deep X ray therapy radium was applied by means of a mouth mould specially constructed for the patient from dental base plate and "Stent". Two teeth in the region of the growth were extracted to facilitate fitting of the applicator. The area treated was nine square centimetres, and the radium used, 22 milligrammes in all, was placed accurately at a distance of 0·5 centimetre from the mucosa. The mould was worn by the patient without discomfort between meals for nine hours during the daytime on successive days, the dose to the mucosa being estimated at 8,000 *r* for the eighty-one hours of application.

At the time of the meeting, three and a half months after the radium treatment, the mucosa over the region where the epithelioma had been appeared normal. There was no scarring, and no evidence of malignant disease was found. There was still no evidence of involvement of the cervical glands.

Dr. Ham pointed out that the result had been achieved without any operative interference whatever. He emphasized the fact that for success with dental radium applicators care had to be taken to choose lesions on the less mobile parts only, and to make sure that the applicator fitted accurately and that the distribution of the radium sources and the dose to the mucosa were accurately worked out. The patient had agreed to attend regularly each month for examination by the surgeon and the radiotherapist, and dissection of the glands would be advised if any developments occurred.

Carcinoma of the Œsophagus.

Dr. Ham's second patient was a male, aged fifty-one years, who was shown to demonstrate the palliative effect of deep X ray therapy in an advanced carcinoma of the œsophagus.

The patient, when first seen at the end of September, 1937, was able to swallow only a little milk. Œsophagoscopy was carried out by Dr. Garnet Halloran on September 24, 1937, and revealed a stricture of the

œsophagus 31·9 centimetres (twelve and three-quarter inches) from the upper incisor teeth. The radiological appearances were those of carcinoma.

The patient was given a six weeks' course of deep X ray therapy, commencing on November 2. Only long, narrow fields were used, three anterior and three posterior, suitably arranged over the thorax. The tumour dose had been calculated at approximately 5,000 *r*. On January 24, 1938, there was improvement in the radiological appearances in the œsophagus; but an X ray film of the chest revealed a large opacity extending outwards into the right lung from the hilum. Further X ray examination on March 31 revealed the œsophagus to be much more regular in outline. There was no obstruction to the barium bolus, and the infiltration in the right hilar region was definitely less than previously. The patient at the time of the meeting was clinically greatly improved, had gained in weight and was able to swallow normally. Dr. Ham was of the opinion that the infiltration in the right lung was probably an inflammatory reaction resulting from the intensive irradiation. This opinion was supported by the time of its appearance and by its subsequent diminution.

Carcinoma of the Tonsil.

Dr. Ham's third patient was a male, aged sixty-two years, who had been treated by deep X rays in August, 1937, for a huge fungating carcinoma of the right tonsil involving the anterior pillar of the fauces and soft palate. No evidence of malignant disease could be detected at the time of the meeting.

Epithelioma of the Lip.

Dr. Ham's fourth patient was a male, aged sixty-three years, who had been treated by deep X rays in November, 1937, for a very extensive epithelioma destroying the right three-quarters of the lower lip and extending to the upper lip and well back into the right cheek. At the time of the meeting, the lesion was replaced by a soft scar, with no clinical evidence of malignancy.

Rodent Ulcer.

Dr. Ham also showed a male, aged seventy-five years, who had been referred to him in January, 1937, with a rodent ulcer of the left inner canthus region and left cheek. Six years previously he had been treated at another hospital, but the lesion had recurred and was rapidly increasing. An implantation of gold radon seeds had been made, the infiltration being through 0·5 millimetre of gold. The seeds were left in position permanently, and at the time of the meeting the lesion appeared healed.

Graves's Disease.

DR. A. T. NISBET showed a male patient, aged thirty years, who had been referred to him on January 5, 1937, suffering from Graves's disease. For the previous six months this patient had been very nervous, excitable and upset by trifles, and had complained that he sweated considerably. For three months he had noticed a swelling over the front of the neck. The basal metabolic rate had been estimated on December 21, 1936, at + 68%. His weight was 57·6 kilograms (nine stone one pound).

A course of small doses of deep X rays to the thyreoid region was begun on January 5, 1937, and continued until March 10, 1937. The patient at the time of the meeting appeared perfectly well. He had gained 25·4 kilograms (four stone) in weight and was carrying on his usual work.

Cerebro-Pontine Tumour.

Dr. Nisbet's second patient was a male, aged thirty-five years, who had been shown at a clinical meeting at the Royal Prince Alfred Hospital two years earlier. In 1931 craniotomy had been performed by Dr. H. R. G. Poate. A tumour had been found at the cerebro-pontine angle. The tumour was considered too deeply placed for investigation and the patient was sent for deep X ray therapy. At that time he was unable to walk, was partially blind, and had headaches, vomiting and aphasia.

Six years later, at the time of the meeting, his eyesight was practically normal. He could read the morning paper, his speech was excellent and he was able to chop wood and perform other tasks as handy-man around a boarding house. Prior to this he had been a jackeroo on a western station.

Subcutaneous Angioma.

Dr. Nisbet's next patient was a male infant who had been referred to him in May, 1937, when twelve months old, with a large subcutaneous angioma of the right cheek, approximately five centimetres in diameter and three centimetres in depth. Four injections of sodium morrhuate had been given previously without any improvement. Two doses of 600 *r* of deep X rays were given with two days' interval between them. A general anæsthetic was administered each time to ensure immobility while the baby was under treatment. The result was excellent; the tumour had disappeared and the skin over it was normal.

Carcinoma of the Breast.

Dr. Nisbet also showed a female patient, aged fifty years, to demonstrate the effect of deep X ray therapy on a very advanced carcinoma of the breast. When first seen in May, 1937, the patient had a large, foul-smelling ulcerated carcinoma, involving practically the whole of the left breast and extending into the axillary tail, the whole mass being fixed to the pectoral muscles and chest wall. Large axillary glands were also present. Microscopic examination of the tumour disclosed extensive involvement by small carcinomatous masses, the cells being mainly cuboidal in type. X ray examination of the chest and spine revealed no evidence of metastases.

The patient was treated by deep X rays, three fields being used. At the time of the meeting the main lesion had disappeared, a soft, healed scar being left. The glands were no longer palpable; but there was a small indurated mass with a few skin nodules surrounding it over the pectoral fold. Dr. Nisbet said that a localized radium implant would seem to be indicated to clear up this area.

Radium and Radon in the Treatment of Malignant Disease.

DR. P. D. BRADDON showed a number of patients with malignant disease who had been treated by radium and radon at the radium clinic.

Dr. Braddon's first patient was a man, aged sixty-seven years, who had presented himself on May 3, 1937, with a rapidly growing epithelioma of the lateral aspect of the left ear of some four months' duration. The lesion was three centimetres in diameter. It was ulcerated and filled the upper half of the concha. In addition, the medial aspect of the ear, exactly opposite the first-mentioned lesion, was occupied by a slowly growing basal-celled carcinoma of twelve years' duration. This lesion was also three centimetres in diameter.

The rapidly growing lesion on the lateral aspect was first treated by a radium mould at a distance of two centimetres. The treatment was continued for ten days, a dose of 6,000 *r* being given. A single circle of needles containing 20 milligrammes was used. The lesion cleared up in a few weeks. On July 7, 1937, all reaction having cleared up for some time, the medial surface of the auricle was treated by a radon mould at a distance of one centimetre for seven days. The total dose was 6,500 *r*. This was obtained by 25 millicuries in 8 needles, each 1 centimetre long and each containing 3·1 millicuries.

By October, 1937, it was noted that an excellent result had been obtained. At the time of the meeting, although the patient had had quite advanced growths on either aspect of the cartilage, it was impossible to detect which ear had suffered malignant disease and been cured by radium.

Dr. Braddon's second patient, a man, aged sixty-three years, had presented himself on December 6, 1937, with a very large, foul-smelling, fungating growth on the dorsum

of the right hand, which bled profusely when handled. The lesion measured 5·5 centimetres in diameter and was two centimetres in thickness.

It was treated by a radium mould at a distance of 2·5 centimetres. The very thick, plateau-like growth itself was used for most of the bulk of the mould, and the dose of 6,000 *r* was worked out for the base of the lesion. For ten days 42·5 milligrammes were used in a single circle. The overpowering odour from the growth was such that the patient had to be put out on the ward verandah during treatment. However, it cleared up so well that on April 4, 1938, it was possible to presage a good result. By May 2, 1938, his card was marked "excellent result". There was no sign of a neoplasm. The skin was almost normal. Full use of the hand was possible and the extensor tendons were free.

Dr. Braddon showed a number of other patients illustrating various phases of radium work. He also showed some moulds and applicators, illustrating methods of working out dosage in terms of γ *r* units, and of obtaining relative homogeneity of the dose from moulds.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the South Australian Branch of the British Medical Association:

Brummitt, Donald William, M.B., B.S., 1938 (Univ. Adelaide), Adelaide Hospital.

Proceedings of the Royal Commission Appointed to Inquire into Matters Pertaining to National Health Insurance.

Friday, October 14, 1938.

The sittings of the Commission in Adelaide were continued at 10.30 a.m. Dr. Neil Melrose Wigg, of Gilberton, a suburb of Adelaide, was examined by Mr. Abrahams. He commenced his evidence by giving confidential information. He gave the figure of estimated diminution of his private practice owing to national insurance as 19%. He gave evidence concerning various hospitals in England in which he had had experience. He stated that in his hospital practice he had seen numerous instances of wrong diagnosis made by general practitioners in England.

Mr. Abrahams: You see on this sheet that we have calculated for you, that to prevent loss, the capitation fee would need to be 18s. I understand that you personally take some view about that?

A.: I do.

Q.: Do you mind expressing it?

A.: I think that that 18s., according to this calculation, only partly recompenses us [himself and two partners] for our loss, because there are several factors which have not been taken into consideration in it. For instance, I think that that figure does not adequately represent a return on our particularly expensive post-graduate experience. We make no claims at all to being more than general practitioners, but actually, in point of fact, we do a great deal of work between us in different spheres, which, in a good many practices, would have to be referred to specialists.

Q.: Is there anything else you wish to say about the aspect I put to you concerning the 18s. we have worked out?

A.: Yes. I think one important factor is that we very carefully examine all lodge patients, both fresh admittances to the lodge and also transfers from other doctors' lists, and we will not accept them unless they are really good risks. That has been our fixed habit, and I am sure that it would entail a considerable amount of extra work attending to the class of patient that I understand will become a national insurance patient.

Q.: On that subject, take the proposal of the Federal Government to initiate a voluntary scheme for the wives and children of insured people; that is not a compulsory scheme at all. Do you think there will be any selection made there by the less healthy dependants? If there is a voluntary scheme, of course, there will be no compulsion upon anybody to come in. I suppose it is fairly obvious that the less healthy, rather than the more healthy, will prefer the voluntary scheme?

A.: Yes.

Mr. Dovey: If I understand you aright, you suggest that this 18s., or whatever the figure may be, is not adequate in your case for three reasons: firstly, it would not represent a return on your particularly expensive post-graduate experience; secondly, because of the compulsory nature of the service, and in that regard you refer to the fact that you are unable at present to cope with all the work that offers at times; and thirdly, that you very carefully examine your lodge patients?

A.: That is so.

Q.: Dealing with the first of those grounds, do you suggest that insured persons who may select you or your partners as their insurance doctors, should be obliged to pay more than if they selected somebody who had had less expensive post-graduate experience?

A.: No. I take it that if we have national insurance we will be obliged to treat them.

Q.: And you know that you need not do it unless you like?

A.: That is so up to a point, but one has to earn an income.

Q.: You have looked at the sum that has been worked out for you by my friend's instructing officers?

A.: I have.

Q.: Have you considered that under national health insurance you would be in a position to charge for and receive payment for excluded services to those people?

A.: Yes, that has been considered.

After further cross-examination upon this subject the Chairman interposed: Is it worth while labouring the matter? The second part of this sheet is compiled by the staff.

Mr. Dovey: Upon an assumption which they make.

The Chairman: It may be right or it may be wrong, but the witness cannot speak as to the justification for the assumption.

Dr. Wigg was then cross-examined upon matters arising out of his confidential evidence and in conclusion was asked by Mr. Dovey: May I take it that your opinion is that the Australian graduate is better trained than his English *confrère*?

A.: That is my opinion.

Q.: How comes it, then, that the Australian graduates so frequently, and at great expense to themselves, go to the halls of learning which turn out these people who are not so well trained?

A.: Because in England I have an admiration for the standard of the special services, but the standard of the general practitioners and the pass standard of the universities is, in my opinion, not so good as here.

Q.: Is that opinion shared by others in Australia, to your knowledge?

A.: It is.

Q.: By your partners?

A.: Yes.

Sir Henry Simpson Newland, of Adelaide, the President of the Federal Council of the British Medical Association in Australia, was called as a witness by Mr. Dovey.

Mr. Dovey: What have you to say on the observation made by the last witness as to the quality of teaching in Australia and England? Do you agree that the English training is not so good as the Australian?

A.: The training in Australia, of necessity, has been of a more wide character, owing to the absence of specialists, more particularly in the outlying districts, so that a man has to be trained, for instance, in diseases of the eye and more special subjects, so as to treat people in the outlying districts. That is not so much nowadays, because means of transport are so rapid that patients can seek the advice of specialists in the cities. From that point of view I would say that the general practitioners in Australia, more particularly in the past, have been trained in those special subjects to a greater extent than the graduates in England.

Q.: Leaving out the special subjects, you heard what the last witness said, referring to the ordinary physician's work, the diagnosis of diseases which any competent man, adequately trained, should be able to make. You heard him express the opinion that mistakes in diagnosis were made by English general practitioners, simple diagnoses, which an Australian graduate would not make because he has been better trained. Do you agree with that?

A.: No, I would not necessarily agree with that. After graduating here I was five years at the London Hospital, and the men there, I consider, are just as well trained as they are here—that is the general practitioners.

Q.: I think you were the President of the executive body which was set up by the Branches of the British Medical Association throughout Australia for the express purpose of meeting the representatives of the Federal Government to discuss the scope for medical service and the rates of remuneration for medical practitioners under the proposed national health insurance?

A.: Yes.

Q.: There were a number of conferences held between representatives of the profession and the Government?

A.: Yes.

Q.: I think you have read a record or notes of their discussions, which appear at page 25 to 29 of the evidence submitted to this Royal Commission by the Insurance Commissioners?

A.: Yes.

Q.: Do you agree that in substance those discussions are set out accurately?

A.: Yes.

Q.: I understand the position was that the Federal Executive, while having no legal authority to bind the profession, felt, and still feels, that it has the moral authority so to do, that is, to speak on behalf of the profession?

A.: Yes.

Q.: You, of course, realized all the time that service under national health insurance was quite voluntary on the part of the individual practitioner?

A.: Yes.

Q.: Some little time after those deliberations came to an end and an agreement was reached, there appeared in The Medical Journal of Australia, of May 14, an article which we have been told was written by the Editor, Dr. Archdall?

A.: Yes.

Q.: We have also been told that the substance of that article was communicated by you to Dr. Archdall?

A.: No, I never communicated the substance to him; I saw the article before it was published in the paper.

Q.: And as President of the Federal Council of the British Medical Association you approved of it as representing the views of the Federal Council?

A.: I thought at the time it represented their views, yes.

Q.: The Federal Council and the Federal Executive?

A.: Well, the Federal Executive, I think.

Q.: I formally tender the extract from The Medical Journal of Australia, which appears at pages 30, 31 and 32 of the evidence of the Commission, as being the express views of the Federal Executive of the British Medical Association in Australia. Your Honour may remember this document has been put in evidence, but this particular article was tendered by me and objected to by my friend, and after some discussion its tender was withdrawn.

The Chairman: You are now re-tendering this particular extract, and apparently it is objected to by Mr. Abrahams?

Mr. Dovey: Sir Henry, speaking as President, says that article represented the views and opinions of the Federal Executive of the British Medical Association in Australia, which was the very body appointed to speak on behalf of the profession to the representatives of the Federal Government on the matters to be inquired into. Its weight is a matter for subsequent discussion.

Mr. Abrahams: Might I be allowed to ask a question of Sir Henry on the *voir dire*?

Mr. Dovey: This is not a court of law, but a Royal Commission of Inquiry, and I have adopted this method as a short cut.

The Chairman: I do not think that is the ground of the objection taken. Sir Henry has not been taken through this in detail.

Mr. Abrahams: No, I want to ask Sir Henry was this document submitted to the whole council or merely to him. If he says it was submitted to the whole council I have no objection to it, but if he says it was merely submitted to him, and as far as his understanding of the matter was concerned it represented the views of the Federal Council, I press my objection. That is why I want the question asked by somebody first of all before I argue it, whether it was in fact submitted to the whole council or the executive of the council, and if it was, then I do not object to it. If it represents Sir Henry's own views of what the opinions of the council might be, then I do object to it.

The Chairman: There is no harm in ascertaining the actual fact of whether it was submitted to the executive, as it has been called, or whether Sir Henry, in effect, approved of it without consulting his fellow members of the executive. If it turns out that it was not submitted, and that, in substance it represents Sir Henry's own views, at present I do not feel inclined to exclude it on that ground.

Mr. Dovey: Did you hear what my friend has suggested?

A.: Yes.

Q.: Perhaps you could tell the Commission?

A.: The article was submitted to me as a suitable article to express what had taken place in our conference with the official representatives. The facts as set forth in that article were correct. The views which were expressed, arising out of those facts, were those of the Editor. They were not my views, but his. I do not say they were divergent from my views, but the article was written by him. It was never submitted to the Federal Executive, but it was approved by me as a suitable article to give an account of the negotiations which had taken place between the executive committee and the official representatives.

The Chairman: And your approval was given, as your personal approval—is that what you mean to suggest—of a statement of what took place at the conference, apart altogether from the Editor's views? Your approval was given as your own approval and not as the approval of the executive?

A.: No, it was given by me as president of the executive.

Q.: Did you consult with any other members of the executive before saying the article was all right?

A.: No.

Mr. Dovey: I may take it you had ascertained the views of the executive on the result of the deliberations at the time of the deliberations?

A.: I was cognizant of their views. Dr. Hunter had also read this leading article before it was submitted to me.

Q.: That is Dr. Hunter who is here?

A.: Yes, I think I am correct in saying that.

Q.: And he had been present at the interview?

A.: He was present at the meeting of the executive.

Q.: Then so far as the article contains statements, they are correct, and so far as they were comments on the facts, they were written by the Editor, but you agreed with the comments, as president of the executive?

A.: Yes.

Q.: I tender the article as being a statement of facts and, comments on the facts which were adopted and accepted by Sir Henry as president of the executive.

Mr. Abrahams: I object on more than one ground. It is a well-known law, and Your Honour will agree with me, taking the case of a company, that it is idle for one director to say, "I knew the views of all the board, I did not have a board meeting; the thing was not decided by a board meeting, but I knew all their views, and this represented their views". The proper way in such a case is to have a board meeting . . . Supposing the whole of the 4,500 practitioners in Australia had met and said to the Government by joint letter, "We think 11s. is the proper rate", and ten minutes later they said, "We go back on it, we will not have it", could not the Government have then said, "Very well, you did not make a binding agreement, you all admitted it was fair, we think it is proper to have a Royal Commission to say what is proper". Anybody could come along later and say they made an admission of what is a fair thing, and that is the thing the Commission must look to, but I submit His Excellency has said to this Commission: "Find out what is the proper fee, because previous discussions that have taken place have led to nothing; find out what is the proper fee, notwithstanding those discussions, and ignoring them."

The Chairman: We think that we should not exclude this evidence of Sir Henry and this extract which is now pending, but in order to prevent misapprehension we should make it clear now that the observations of Mr. Abrahams concerning its value will probably be regarded by us as being of enormous weight when we are considering finally how this piece of evidence should be treated. It may be that although we admit it now, ultimately we will put it aside altogether and not allow it to affect our decision, but at the same time we think it should be admitted in that tentative fashion.

Mr. Dovey: I know the Commission will allow me an opportunity at the proper time to be heard on their ruling.

The Chairman: In our opinion, what Mr. Abrahams has said is matter which may be urged with very great force in Mr. Abrahams's final address, but at the present time we think we should not exclude this tendered evidence, and it should be admitted.

Mr. Dovey: The weight of it as evidence will be discussed later?

The Chairman: Yes, it might be put aside as of no value for our purposes.

Mr. Dovey: You remember after the deliberations had come to an end, there were meetings in several States?

A.: Yes.

Q.: Some members of the profession thought the rate was not sufficiently high?

A.: Yes.

Q.: As a result of that, were representations made to the Federal Council?

A.: Yes.

Q.: The Federal Council realized it could not legally bind anybody in the first place?

A.: That is so.

Q.: After representations were made to the Federal Council by the different Branches, the suggestion was made in a letter of May 21, appearing at page 32 of that report, that the minimum rate acceptable would be 14s. in the metropolitan areas, with an additional 25% for country areas?

A.: Yes.

Q.: That letter concluded with a request to the Federal authorities for a further consultation?

A.: Yes.

Q.: As far as you know, that letter was not answered by letter?

A.: No.

Q.: On June 13 another letter was written, apparently on behalf of the Federal Council of the British Medical Association, which said this, amongst other things . . . ;

> The terms contained in our letter to you of 21st May would be acceptable to our members.

Do you remember that?

A.: Yes.

Q.: Do you know on whose authority that statement was made?

A.: I presume it was made on the authority of the council itself.

Q.: Is it a fact that by that time the Federal Council of the British Medical Association had ascertained that the terms suggested in the letter of May 21 would be acceptable to the rank and file members, the general practitioner members of the Association?

A.: Inquiries have been made in the different States to ascertain what the views were, and I think it is correct to say that it was thought that those terms would be acceptable to the profession. They had no more authority to say so.

Q.: But the Federal Council had been assured by the different Branches that those terms would be acceptable to the different Branch members?

A.: I do not know that we have documentary evidence to show that each Branch had accepted them, but that was the general tenor.

Q.: Large numbers of the profession were urging that the profession should have nothing whatever to do with national health insurance?

A.: Yes.

Q.: Speaking broadly, you think those terms would have been acceptable to a lot of the members?

A.: Yes.

Mr. Abrahams: "To the Branches", he said, not the bulk of the members.

Sir Henry Newland was then cross-examined by Mr. Abrahams.

Mr. Abrahams: I take it your council had no authority to bind the whole profession?

A.: No.

Q.: And you did not purport to do so?

A.: I think at the time we assumed we had the authority to bind the profession.

Q.: But it was made clear to the official representatives—right at the early stages there was some discussion of this sort, "We cannot get our members to accept anything of that nature"—you had to go back to your profession?

A.: We distinctly said that when we went back; that is how our conference broke up, on that very point.

Q.: That is to say, you did not purport to bind them?

A.: No.

Mr. Dovey: When you say you thought you could bind them, you meant you felt that your executive, having been appointed by the profession as a whole for a particular purpose, if you did your job to the best of your ability and arrived at what you regarded as a fair agreement your actions would be honoured by the profession which had appointed you?

The Chairman: "Honoured" is hardly the word.

Mr. Dovey: It would be "confirmed".

The Chairman: That what you recommended would be accepted by the general body of medical practitioners?

A.: We thought that might be accepted, but as I specifically said at the conclusion of the negotiations, the agreement was accepted by the Federal Executive, but we could not bind our members because they had not had the opportunity of considering them.

Q.: You had been appointed to negotiate, and you expected what you had recommended would be accepted by the general body of practitioners, not as a binding agreement upon them, but on the whole, as something which they could adopt?

A.: We thought so at the time, at the conclusion of the conference. We were very careful to say that we could not bind our members.

Dr. Stewart Roy Hecker, of Brighton, a seaside suburb about nine miles south-west of Adelaide, was examined by Mr. Gain. Dr. Hecker gave evidence of a confidential nature concerning his private and his lodge practices. He estimated that his services to private patients would diminish by 29% as a result of national insurance. Dr. Hecker was then cross-examined by Mr. Dovey on these matters and then withdrew.

Dr. Hugh Selby Covernton, of Port Adelaide, was called as a witness by Mr. Dovey and was examined by Mr. Bowie Wilson. Dr. Covernton gave confidential information concerning the lodge practice conducted by himself and his partners, and also concerning their private practice. He was cross-examined on these matters by Mr. Abrahams.

Dr. James Edward Everard, of Colonel Light Gardens, was called as a witness by Mr. Dovey. He described Colonel Light Gardens as a district inhabited by the poorer paid labouring classes. Dr. Everard gave evidence of a confidential nature concerning his lodge and private practices.

Mr. Dovey: Have you thought how national health insurance might affect your position?

A.: I do not think I will be so well off.

Q.: How is that?

A.: I have formed the opinion; I cannot say why. For one thing I think my lodge practice will go down.

Q.: You mean the lodge members who are individuals will cease to be lodge members and will come under national health insurance?

A.: Yes, I think most of the females will go off. I do not know what is going to happen.

Q.: Do you think so because you have been told so by people?

A.: I have asked the people and they say they cannot afford the £3 18s. a year as lodge fees. They are wondering what is going to happen.

Q.: I suppose they are wondering whether there will be any reduction in their lodge contribution?

A.: I suppose so.

Mr. Gain: One definite way you have in mind that you might lose is that people who are now dependants of lodge members will, when those members come under the Act, cease to be covered by a lodge?

A.: Yes.

Q.: That is because the members themselves fear that they will not be able to pay anything at all in lodge fees for their dependants if they have to pay £3 18s. into national health insurance?

A.: Most of my members out there are on the basic wage.

Q.: One shilling and sixpence per week would be a very serious matter to them?

A.: To most of them.

Q.: In answering Mr. Dovey you said you were of opinion that the excluded services were more valuable to you than the included services, and you had in mind particularly minor operations with a local anæsthetic?

A.: Yes.

Q.: That is the most profitable part of your practice?

A.: Yes.

Q.: It was because of that item that you were of opinion that the excluded services were more valuable?

A.: Yes.

Dr. Herbert Henry Ernest Russell, of Unley, a suburb of Adelaide, was called as a witness by Mr. Dovey. He described Unley as a middle-class residential suburb. Dr. Russell gave confidential evidence on similar lines to the other witnesses.

Mr. Dovey: You have had many years' experience of contract patients?

A.: I have had lodge patients from the first year I was out there.

Q.: And your relations with the lodge patients and with the lodge people are most harmonious?

A.: Yes.

Q.: I think that your words to me were that you would just as soon have your lodge patients as your private patients?

A.: Yes.

Q.: Do you find that your lodge patients give you much trouble in calling you out unnecessarily?

A.: No, I do not. They come to me chiefly.

Q.: I think that you are able to say to the Commission, over many years' experience, what is the practice of lodge patients contrasted with private patients in coming to the doctor?

A.: When a private patient becomes a lodge patient he sees the doctor ever so much more. There are several reasons for that. A private patient with influenza might not come to the doctor at all. Once he is a lodge patient he wants to go on the funds of the lodge. Therefore, he has to go and see the doctor to put him on the funds, and he has also to go and see the doctor to take him off the funds. There are two visits which he would not have had if he had been a private patient. Then again, you tell a private patient, who has, we will say, anæmia, "Here, take six bottles and come back". A lodge patient you tell the same. The private patient does not come back till the end of the six bottles, but a lodge patient will come back for every repeat. Some lodges will allow us to give two, which means that the lodge patient is making three visits to the other's one. Those are the two chief reasons, I think.

Q.: You think that lodge patients come at least twice as often as private patients?

A.: I think they do.

Q.: I do not know whether you considered or made any estimate of the percentage of your private patients who would come under national health insurance?

A.: My brother said quite 60% of his would, but I do not think any more than 50%, if 50%, of mine would.

Q.: You will appreciate it is only the employee, not the dependants, who come under the national health insurance. You mean 60% of the wage-earners would come under it?

A.: Yes, of the wage-earners. The women come under that, do they not?

Q.: If they are employees. Your figure is very high?

A.: I have not gone into that thoroughly.

Dr. Russell was cross-examined by Mr. Gain, mainly upon figures that he had given, and he then withdrew.

Dr. Douglas Robson Wallman, of Hindmarsh, a working-class suburb of Adelaide, was called as a witness by Mr. Dovey. He gave confidential details with respect to his lodge practice. He was cross-examined on these matters by Mr. Abrahams and withdrew.

The Commission adjourned at 3.40 p.m. until Monday, October 17, 1938, at 9.30 a.m., at Kalgoorlie, Western Australia.

Monday, October 17, 1938.

The sittings of the Commission at Kalgoorlie, Western Australia, opened at 10 a.m. on Monday, October 17, 1938.

Mr. Dovey: If Your Honour pleases, it has not been possible for us to call any witnesses here, but we hope through the Press, this being our first sitting in this State, for the benefit of those people in Perth, to notify them that we will welcome any person or organization who think they may be in a position to put evidence before the Commission to assist them in the inquiry.

Mr. Abrahams: There are two or three matters that have to be dealt with here specially. The first one is the system of contract outside of the lodges which prevails in the mines here, as it does in other big mining centres in Australia. At Broken Hill and Newcastle, New South Wales, there is a contract practice by which, for a lump sum paid through the office of the mines, attention is given to the miner and his family. There is, however, a marked difference between the contract here for the miners and their dependants and the contract prevailing in New South Wales. The position here appears to be that a sum of 3s. each pay for twenty-four pays in the year, amounting to

£3 12s., is deducted from the miner's wages, and for that he and his dependants get the following attention: they all get the included service included in the lodge agreement, with which we are familiar in other States. The lodge agreement here is somewhat similar as regards service to that in force in New South Wales. They all get the included lodge service, exclusive of confinements, and so on. They also get medicines, which come out of the 8s. or 9s. a year per member. In addition to that, the member only, and not his wife or children, is entitled to free hospital service, and he is also entitled to operations of necessity. There seems to be a divergence of opinion as to what are operations of necessity. One can think of operations which are not operations of necessity, such as a wart on a man's foot, or some minor thing of that sort. There are also more serious matters which are not operations of necessity, such as a hernia, which is not a strangulated hernia, where an operation is performed according to the election of the individual and for his comfort, as distinct from his safety. There are those cases to which attention has been called, like an appendix, where a medical man says something like this: "It might not trouble you again, and you can perfectly safely wait; there is no need to have it out. If you take my advice you will have it out now and be done with it, because I think it will trouble you again, but it may not; please yourself what you do." One gentleman at the table has had that very experience recently. For the most part one would have thought major operations were operations of necessity, but that does not seem to be the established custom here, because the doctors have informed us that under that practice they do very few operations.

There is a large practice in Kalgoorlie in workers' compensation. They have the miners and wood-cutters who cut all timbers for the mine, and there is a very big field. There are some operations which come under the *Workers' Compensation Act*, but for the most part those operations of necessity, as far as one can gather from a consensus of opinion, are very few. The result seems to be that this contract system, existing in the mines and amongst the wood-cutters, is a much more beneficial one for the doctors than the contract practice, which is at the rate of 26s. in this city. That is the usual contract rate, family rate, and single men pay the same. I am informed there are a great number of single men in this district, miners and wood-cutters. I am proposing to call first a doctor who attends to about 300 wood-cutters, and there are only 40 of them who have families. There is a large proportion of single men in this area, and the fee is 26s. It is important to notice that the contract fee in Perth is 24s., and here 26s., which by no means makes up for the difficulties in the way of the Kalgoorlie practitioner. I am not referring to the amenities, but the cost of living. We are told that petrol in Kalgoorlie costs 2s. 8d. per gallon, whereas the Perth price is 1s. 9d. Living generally is much more expensive in Kalgoorlie than it is in Perth, and there does not appear to be that parity which should exist between Kalgoorlie and Perth. Nevertheless, perhaps some compensation exists in the fact there is a large mining population. I think the population of Kalgoorlie and Boulder is about 25,000, and of those, 3,600 are miners, that is, without dependants. There are a great many workers also whose livelihood depends on the continued existence of the mines.

One witness I am proposing to call, at first sight may appear to give evidence which cuts, to some extent, against the case of mileage so far made by the British Medical Association. I am calling him, however, for two reasons: firstly, because we are desirous that the Commission should get a fair sample throughout Australia; and secondly, it does show the fallacy of this scheme propounded by the Insurance Commission that mileage should be paid at *per capita* instead of on the distance travelled. If mileage was paid to this doctor for 300 men working seventy miles away, timber-cutting for the mines, he, being the nearest doctor, would receive £2,100 a year for mileage, which is absurdly high. He would receive £7 per man *per annum*, and there are 300 employed there permanently—it is not a mere timber-getters' camp. They are seventy-two miles away at present, and the distance changes according to the work they are doing. These men have been there for about seven years, and they are likely to go on as long as the mine or the timber lasts, and it may mean that this doctor, who goes out there once a week at a cost of £3 for his transport—he has to hire a sort of trolly to get there, because the roads are so bad—if the Commission's scheme were carried out, this gentleman would receive £2,100 a year for mileage, which is absurd. We have said all along that mileage should be paid according to the distance actually travelled.

Mr. Dovey: You do not suggest that is a typical case?

Mr. Abrahams: In the other cases of the normal practitioners it works great hardship. This only throws into relief the fallacy of applying to this country conditions which exist in England, and it shows the proper way to apply mileage is to the distance actually travelled.

Mr. Dovey: It would be helpful if my learned friends, at some convenient time, could formulate a plan which, in their opinion, would work satisfactorily in practice and at the same time be within the legal powers of the Insurance Commissioners.

Mr. Abrahams: We have suggested the doctors should be paid by the patient as at present and that there should be a clause in the contract to that effect. His Honour made the suggestion first, which we adopted, that there should be a clause in the contract about mileage, and it should be at the same rates as exist under contract practice at present, namely, 5s. per mile one way over two or three miles. I do not bind myself to that; in some States it is two miles and in others three miles.

Mr. Dovey: That does not accord with the legal interpretation that my friend puts on Section 47.

The Chairman: I think we will have more time to devote to this in Melbourne or Sydney than here.

Mr. Abrahams: Another matter is that from Perth a census has been attempted, somewhat on the lines of that carried out in Victoria, and the doctors in these towns have contributed, or at least six that I know of. The method adopted has been to ask the lodge secretaries to fill in the information, and when we get to Perth I shall give the tabulated results. Here I will merely attempt to show what has been done in this city in regard to a census by the secretary of one of the doctors who had the handling of a fair proportion of what was done here, and I propose to call her. One or two of the doctors who will be called this morning with regard to figures are also concerned with the census, and I would ask the Commissions' indulgence to allow Mr. Gain to examine the witness on the figures aspect, which I have not had a chance to prepare, and allow me to examine the same witness on the census aspect.

Dr. Morris O'Connell Gorman, of Kalgoorlie, was examined by Mr. Abrahams. He gave detailed evidence concerning the question of mileage in relation to the timber-cutters' camp mentioned by Mr. Abrahams in his opening. Dr. Gorman also described the operation of the mines agreement and general conditions in the district.

Dr. Aloysius Daly Smith, of Kalgoorlie, was examined by Mr. Gain. Dr. Smith gave a description of the hospital accommodation and general living conditions in Kalgoorlie.

Mr. Gain: What is the nature of the contract practice in Kalgoorlie?

A.: There are three types of contract practice. The first one is the Mines Medical Fund, then the friendly societies practice, or lodge agreement, and then there is the Medical Trust Fund. The Mines Medical Fund is a condition of employment of the mines whereby each person employed not being on the staff of the mines has 1s. 6d. deducted from his weekly pay. That is paid into the Mines Medical Fund and the mine sends us monthly cheques with the names of those men. The choice of doctor is free. Every doctor on the goldfields is a member of this Mines Medical Trust, and the choice is free to the patient. The money is paid from the mine to the doctor with a list of those patients or members on his list for that particular month.

Q.: You are paid at the rate of 6s. a month?

A.: Yes, £3 12s. a year.

Mr. Dovey: Paid in advance?

A.: No, usually six weeks afterwards. If it was the month of July, we would get our money in the middle of August, and the list.

Mr. Gain: What service do you render for that under the Mines Medical Fund?

A.: That covers a member and his wife and family for attention and medicine, and himself for hospital.

Q.: What about operations?

A.: There is a clause in the agreement which says that operations of necessity are not to be charged for. Operations of election are excluded from the contract.

Dr. Smith described the operation and extent of the Mines Medical Fund and was then asked by Mr. Gain: That covers the medical fund, but within the lodges are they the ordinary contract practices?

A.: They are not really, because the lodges here are fairly large. A mine worker can pay into the medical fund or be a lodge member, and if he decides to be a lodge member the money is deducted at the mine and paid to the lodge and we receive the capitation fees, but it makes the lodge more numerous than they would be otherwise.

Q.: It does not differ from an ordinary lodge contract practice otherwise?

A.: No.

Dr. Smith gave a description of the Medical Trust Fund and its operation, and then gave confidential evidence relating to his practice and certain returns which he had furnished. He was cross-examined by Mr. Dovey on these matters and then withdrew.

Miss Verna Ray Miles, secretary to Dr. Thomas Patrick Byrne, of Kalgoorlie, was examined by Mr. Abrahams.

Miss Miles gave evidence concerning the filling in of the *questionnaire* sent to Dr. Byrne and as to the nature of Dr. Byrne's practice.

Dr. Allan Bickford Webster, of Kalgoorlie, was examined by Mr. Gain. Dr. Webster gave evidence of a confidential nature as to his practice and the answers given by him in the census conducted by the Western Australian Branch of the British Medical Association. He was cross-examined by Mr. Dovey on these matters and as to the operation of the mines agreement.

Mr. Dovey: Do you think there is any confusion about what is an operation of necessity?

A.: Yes, there is always a lot of confusion. For one thing, the definition has never been established of what constitutes an operation of necessity or one of election. Patients have gone into hospital for an operation which a doctor would call an operation of election, and afterwards the patient would claim it to be an operation of necessity. It has not occurred in my practice, but I know it has occurred here.

Q.: What happens then?

A.: The doctor does not collect his fee.

Q.: You know that under the agreement there is provision for the setting up of a board of reference, consisting of a representative of the medical officer, the employer and the subscriber. Advantage has never been taken of that provision?

A.: I have never known of it.

The Chairman: I suppose if a doctor did make a protest and required a board to be set up he would do so at the risk of considerable odium?

A.: He certainly would lose by it.

Q.: In a place like this, and he prefers to lose the money rather than incur that bad feeling?

A.: Exactly.

Q.: So that a clause like that is a mere empty thing.

Further evidence was given concerning the nature of an operation of necessity and as to the taking of the census, and the witness then withdrew.

The Commission adjourned at 12.30 p.m. until Wednesday, October 19, 1938, at 10.30 a.m., at Perth.

Medical Practice.

THE KOCH CANCER TREATMENT.

It is likely that many medical practitioners will receive inquiries about the Koch cancer treatment, as this method has recently been the subject of such widespread publicity in the lay Press. For the information of medical practitioners we should mention that Koch's "Glyoxylide" has been in use for some twenty years. It is used not only in cancer, but also in tuberculosis, psoriasis, asthma and other chronic conditions. An investigation of Koch's method was conducted by the Bureau of Investigation of the American Medical Association; the results were published in *The Journal of the American Medical Association* of August 15, 1936, at page 519. The following quotations from this report will serve to show that it was unfavourable:

> . . . there is nothing even slightly resembling scientific evidence to justify any confidence in its merit . . .
>
> Undisputable records do not reveal a single instance in which a case of unquestioned malignant disease has been cured by the Koch treatment.

William F. Koch graduated from the Detroit College of Medicine and Surgery in 1918.

Correspondence.

A CONSIDERATION OF GENERAL ANÆSTHESIA FOR DENTAL SURGERY.

Sir: Dr. Noel Heath's paper, "A Consideration of General Anæsthesia for Dental Surgery" (The Medical Journal of Australia, Volume II., Number 15, page 594), raises a problem which can be solved only by mutual cooperation between the medical and dental professions.

Pulmonary sequelæ are far from uncommon after dental surgery under general anæsthesia. Open ether in the dental chair or in the patient's home should be completely banned. Who can foretell that an operation will take only a certain few minutes? Reinduction of anæsthesia, partly through an operation, with blood *et cetera* in the mouth, is inviting trouble.

There are certain methods of anæsthesia which render these operations practically safe.

1. For minor dental surgery safety is conferred by the nasal administration of nitrous oxide and oxygen under pressure. After induction the mouth is opened and packed with gauze in such a way as to prevent mouth-breathing and the inhalation of blood or fragments, but to still allow free nasal respiration. The packing is to be renewed as and when necessary.

2. For all major dental surgery complete safety is conferred only by the use of the endotracheal inhalation method of anæsthesia, in which a large-bore catheter is passed into the trachea, and the pharynx firmly packed with gauze. In this way the respiratory system is isolated from the mouth and nose. Ether or one of the gaseous anæsthetics may be used. There is no need for haste in operating. It seems very difficult to convince the dentist of this, as he is still under the influence of the "smash and grab" method, where haste was necessary and added to the danger of the operation. In endotracheal insufflation anæsthesia, in which a small catheter is passed into the trachea and the anæsthetic is delivered at a positive pressure, reliance is placed on the outflow of air through the larynx to prevent the entry of foreign material into the bronchial tree. This method confers relative rather than absolute safety. Unless properly carried out, it may enhance the possibility of foreign material entering through the larynx.

In a dental operation the dentist is the surgeon in charge of the patient, and it is his right to demand certain standards of anæsthesia for his work. This he can obtain only by refusing to work under conditions falling short of this standard. If the patient's usual medical attendant cannot supply the required service, it is in the interests of the patient's welfare to call in someone who can do so.

Yours, etc.,

Douglas G. Renton, M.B., B.S.

12, Collins Street,
Melbourne,
October 10, 1938.

FINAL REPORT OF THE COMMONWEALTH ADVISORY COUNCIL ON NUTRITION.

Sir: Under the caption "Special Abstract: Final Report of the Commonwealth Advisory Council on Nutrition", appearing in the issue of October 8, the following words appear on page 614: "In at least 25% of the dietaries studied, the quantity of milk consumed was definitely below the minimum amount necessary for adequate nutrition."

Would whoever is responsible for the promulgation of the idea of the necessity of milk be good enough to offer us further enlightenment? The list of distinguished names set down as comprising the council is assurance that the term is not being loosely used.

The reason I ask this is that in *The British Medical Journal* of August 20 last there appeared a contribution by Sir Leonard Hill which strongly suggests that milk is not a necessity in childhood at any rate. Sir Leonard, in an article headed "A Strict Vegetarian Diet", instances a superbly healthy boy of nine years who "eats no meat, fish, eggs or bread at home, and scarcely any milk or cheese, but is allowed to eat bread and butter and cake when he goes out to tea about once a week".

That there is convincing evidence that milk is a valuable dietary component seems obvious. But what is the evidence that milk is necessary in post-suckling life?

I feel sure that there are many like myself who would welcome enlightenment.

Yours, etc.,

Sydney, J. A. R. Murphy, D.P.H.
October 12, 1938.

NATIONAL HEALTH INSURANCE.

Sir: For some time now we have seen ourselves being governed more and more by regulations. In the many letters you have printed on national health insurance I do not think this very serious aspect has been sufficiently stressed. From what we read of the Commission now sitting it would appear that the regulations governing the insurance Act are considered as hardly worth discussion. I very much doubt if half the regulations of the *National Health Insurance Act* are even known to the politicians who rushed the Act through Parliament.

Mr. Brigden has now given out that hospitals can refuse to admit patients sent for admission by national insurance doctors.

For many years the rule in the country has been that, except in the case of accidents, a patient must have an admission order from a doctor before being admitted to hospital.

We can now expect that in the country the local hospital secretary will decide what patients shall be admitted. National insurance doctors will find as time goes on that by fresh regulations additional duties, both medical and clerical, will be thrust upon them.

If we still value our liberty, now is the time for our profession to say we refuse to work under soul-destroying regulations.

Dr. Joad (University of London) has written: "We must not give men irresponsible power, not only because it corrupts them and they abuse it, but also because they do not experience the effects of their use of it; they do not, in other words, have to live under the laws they make."

Yours, etc.,

Glen Innes, A. J. Corfe.
New South Wales,
October 13, 1938.

Sir: The suggestion made by Dr. Michael Kelly, of Bunbury, that a plebiscite be held to determine the willingness of practitioners to accept service under the national health insurance scheme, is an excellent one. Time is going on; the Government is pushing ahead with its plans for the introduction of the scheme in January next, a bare eleven weeks away, while the profession follows the report of the proceedings of the Royal Commission and hopes for the best.

The scope of inquiry of the Royal Commission is limited to the question of the remuneration to be paid to insurance doctors, it being accepted by the Government that the scheme itself meets with the approval of the profession, a belief which correspondence on the subject by no means supports.

The profession has already declared, in no uncertain terms, that it will not accept a capitation rate of 11s. *per annum*. If the Commission should find that 11s. be a fair figure, presumably the profession will reject it. If, on the other hand, the Commission recommends 14s., say, it is very doubtful whether the Government will accept it. The Federal Treasurer has stated more than once that he will not accept anything which might upset the actuarial basis of the scheme.

Moreover, the insurance experts who presented the scheme to our Federal Council, stated that if practitioners would not accept a capitation rate of less than 14s. it would be impracticable to include medical benefits in the scheme.

It is difficult to see, therefore, what useful purpose the Commission can serve.

Let our Association put Dr. Michael Kelly's suggestion into effect throughout the Commonwealth immediately. We will then know where we stand.

Yours, etc.,

Wyong, J. H. Rickeard.
October 15, 1938.

Analytical Department.

"OLIO SASSO."

"Olio Sasso" is a brand of olive oil manufactured in Italy and imported by Messrs. John Callose and Sons. A sample was submitted to our analyst, who reports as follows:

We have examined the sample of "Olio Sasso" olive oil submitted by you and found as follows:

	Sample.	Required by British Pharmacopœia (1932).
Specific gravity at 15·5° C.	0·917	0·915-0·918
Refractive index at 40° C.	1·4620	1·4605-1·4635
Acid value	0·69	Not more than 2·0
Saponification value	190·0	190-195
Iodine value	83·6	79-88

The sample complies with the tests for the absence of cottonseed oil, of sesame oil, and of arachis oil.

On this evidence "Olio Sasso" can be recommended with confidence wherever the prescription of olive oil is deemed advisable.

Diary for the Month.

Nov. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 2.—Western Australian Branch, B.M.A.: Council.
Nov. 3.—South Australian Branch, B.M.A.: Council.
Nov. 4.—Queensland Branch, B.M.A.: Branch.
Nov. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 9.—Victorian Branch, B.M.A.: Branch.
Nov. 11.—Queensland Branch, B.M.A.: Council.
Nov. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 16.—Western Australian Branch, B.M.A.: Branch.
Nov. 17.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 23.—Victorian Branch, B.M.A.: Council.
Nov. 24.—New South Wales Branch, B.M.A.: Branch.
Nov. 24.—South Australian Branch, B.M.A.: Branch.
Nov. 25.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. R. E. McClelland has been appointed Government Medical Officer at Stroud, New South Wales.

* * *

Dr. A. J. Corfe has been appointed Government Medical Officer at Glen Innes, New South Wales.

* * *

Dr. R. F. Baird has been appointed Government Medical Officer at Norfolk Island.

* * *

Dr. L. A. A. Forbes has been appointed Government Medical Officer at Woodburn, New South Wales.

* * *

Dr. G. C. Smith has been appointed a Medical Officer in the Office of the Director-General of Public Health of New South Wales.

* * *

Dr. E. C. Egan has been appointed a Senior Medical Officer in the Department of Mental Hospitals of New South Wales.

* * *

The undermentioned have been appointed, in pursuance of the provisions of *The Workers' Compensation Acts*, 1916 *to* 1936, of Queensland, Medical Referees for the purposes of those Acts: Dr. A. V. Meehan, Dr. O. E. J. Murphy, Dr. W. L. Gibson, Dr. E. J. McGuinness, Dr. H. W. Savige, Dr. A. E. Lee, Dr. B. B. Barrack and Dr. F. J. Booth.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii to xxi.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Junior Resident Medical Officer.
HEATHERTON SANATORIUM, CHELTENHAM, VICTORIA: Assistant Medical Officer.
INNISFAIL HOSPITALS BOARD, INNISFAIL, QUEENSLAND: Assistant Medical Officer.
LORD HOWE ISLAND BOARD OF CONTROL: Medical Officer.
MATER MISERICORDIÆ PUBLIC HOSPITALS, BRISBANE, QUEENSLAND: Resident Medical Officers.
QUEEN'S MEMORIAL INFECTIOUS DISEASES HOSPITAL, FAIRFIELD, VICTORIA: Consultant Surgeon.
ROYAL HOBART HOSPITAL, HOBART, TASMANIA: Resident Medical Officer.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officers.
VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Medical Superintendent.
WESTERN AUSTRALIAN PUBLIC SERVICE: Junior Medical Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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